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Title.

OBSERVATIONS ON THE ANATOMY AND PATHOLOGY OF THE
RENAL AND CARDIAC BLOOD VESSELS.

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RENAL BLOOD VESSELS

PART ONE.

Observations on the anatomy and pathology
of the
Renal Blood Vessels.

OBSERVATIONS ON THE PATHOLOGY AND ANATOMY OF THE
RENAL BLOOD VESSELS.

This work was undertaken to classify and study the blood vessels of the Kidney, and to find out what percentage of kidneys have accessory blood vessels.

The normal state as pictured in most books, is a pair of kidneys each with its one renal artery passing from the Aorta to the pelvis.

Is this the state of affairs in the majority of cases, or is it the state in the minority?

What reserve of blood supply has the kidney; what is the embryology of the vessels; what part do the vessels play in high blood pressure; what part do they play in the production of hydronephrosis and infarcts; are the vessels prone to calcification; what are the relations of the renal vessels to those of the suprarenals from the point of view of the hormonal and physiological control of blood pressure?

These are some of the many important questions to be answered from a study of the renal arteries.

Some of my observations from the study of several hundred kidney systems are set down below.

Observations on (A) The work of Goldblatt and others.

Great importance has been put on the amount of blood going into the kidney in relation to the production of ischaemic and hypertensive states.

Briefly Goldblatt and others produced experimental hypertension in dogs by partial occlusion of the renal artery causing an ischaemia.

Goldblatt H. 1937. Ann Int Med II.69.

" " 1937. J. Exper Med. 65. 671.

" et alia 1937. Ibid. 65.233.

" and Kahn 1938. J. Am. Med. Assoc. II0.686.

" et alia 1932. Bull Acad Med Cleve. 16. 6.

" et alia 1934. J. Exper. Med. 59.347.

" et alia 1941. Ibid. 73. 439.

" and Wartman. 1937. Ibid. 66.527.

Hartwich A. 1930. Zischr f d ges exper med. lxix. 462.

It may be noted that this work was done on dogs, which do not from many dissections I have performed have so outstanding an accessory supply as man.

Even in dogs after partial occlusion of the renal artery the establishment of collateral circulation will in time ^{as} lead to amelioration of the hypertensive symptoms.

A dogs kidney has branches of the renal artery which via the capsule anastomose with the lumbar arteries. If the renals are tied, and the aorta injected with radio opaque fluid, the renal vessels can be filled per the lumbar.

The photographs in the following pages show that the accessory supply of the human kidney is in a great percentage of cases very strong, and the possibility of collateralisation in the event of a vessel being blocked, very great.

In dissections of many cases I have found main or accessory vessels almost completely blocked by pathological conditions yet the patient had no hypertension, even when it was evident that the obstructive conditions had been present for a considerable time. This has also been noticed by the undermentioned.

Monakow P. von. Arch f Klin Med 1920. cxxxiii. 129.

Bausi H.H. Zur Hypertonufrage Klin Wehnschr. 1925. 409.

Kimmelstill P. Virchow Arch f Path Anat. 1933. ccxc. 245.

Regarding the mechanism of hypertension production, so far, the following points seem to have been made out. Hypertension of the type referred to does not seem to be of primary nervous nature, as it occurs

(1) after denervation of kidneys.

Goldblatt. J. Exper Med. 1934. lix. 347.

Goldblatt. Ann Int Med. 1937. xi. 69.

Collin D.A. Hypertension in denervated kidneys.

(2) after sympathectomy.

Freeman N.E. Hypertension in sympathectomised dogs after constriction of renal arteries.

Am Heart Jour. 1937. xiv. 405.

(3) after destruction of cord.

Glenn et alia. Experimental hypertension in dogs after destruction of spinal cord.

Amer J. Phys. 1938. cxxiv. 106.

(4) even after kidneys isolated and connected with body by cannula.

Enger R. et alia. Ztschr f.d. Ges Exper Med. 1938. cii. 413.

But the central vaso motor system must play some part because pithing abolishes the hypertension produced.

Dock W. Amer Jour Phys 1940. cxxx. 1 to 8.

Most authorities agree that the vasopressor substance is in the kidney.

Wolf H.J. et alia. Arch f exper Path u Pharmacol. 1935. cixxix. 15-23.

Harrison T.R. et alia. Proc Soc Exper Biol and Med. 1936. xxxv. 38-40.

Prinzmetal M. Pressor effect of kidney extracts from patients and dogs with hypertension.

Proc Soc Exper Biol and Med. 1936. xxxv. 122.

It does not seem to be quite clear yet as to the mode of production of the vaso pressor substance. Is it from granular cells in the walls of the juxta-glomerular portion of the afferent arterioles.

Goormaghtigh H. et alia. Proc Soc Exper Biol and Med. 1939. xlii. 227.

1939. xliii. 688.

Dunihue F.W. Arch Path 1940. xxix. 777 to 784.

What is the role of the Suprarenal gland? Goldblatt showed in 1937 that when both adrenals are removed and the animals are maintained on Sodium Chloride and Sodium Bicarbonate, it is impossible to obtain sustained hypertension by the partial occlusion of the renal artery. The adrenal cortex and not the medulla is the essential part for the production of experimental hypertension.

Goldblatt's papers mentioned before.

Recent Advan Pathology. Garrod. Hadfield. 4th Edit. 1942. 187.

In view of the above summary of recent work on the pathology of obstructive and experimental hypertension the following points are noticed in dissections of specimens and in studying the photographs,

(1) There is evidence of a rich accessory supply in human kidneys.

(2) The human renal vessels have a rich nerve supply and intimate neural connections between them and the coeliac ganglion and suprarenal nerves. See the photograph on Page four and the microphotographs on pages five and six. The vessels of dogs have the same close nerve connection and rich vasculo-neural supply, so in so small an animal it is impossible to clamp vessels without interfering with nerves.

(3) As shown in photographs on pages 14, 22, and 23 etc there is also much vascular connection between the upper pole of the kidney and the suprarenal gland. It must be remembered that the vessels shown in the photographs are only the larger ones that it is possible to dissect without tearing. Close examination under the suprarenal with a magnifying glass show many delicate neural and vascular connections. This confirms the importance of the suprarenal in its team work with the kidney in the control of vascular tension.

- (B) Observations on the embryology of the renal vessels.

In Human Embryology 1933 Arthur Keith states that the definitive arteries of the kidneys are derived from those of the second lumbar segment, and that frequently more than one pair persist. Statistically from the work of others, and from my own figures, (page 51a) the paired accessory arteries are in the minority- 25% as compared with 75% unilateral.

Keith also states in the same work that originally the renal buds at their first appearance receive temporary branches from the Common Iliac and Aorta, but when they come to lie on the dorsal aspect of the Wolfian body in the seventh week, an arterial network which supplies that body and its tubules invade the nephrogenic tissue of the renal buds, and thus the kidney annexes the series of Wolfian arteries stretching from 11th Thoracic to 4th Lumbar.

Jeidell H. 1911. Anat Rec. Vol v. 47 to 54. and Evans H.M. 1912. Keibel. Embryology Vol 2. 570. state that the kidneys are organs which make extensive migrations during growth from one position to another, and many retain vessels from their original location, or receive or incorporate vessels of their new region.

Broman I. 1906. Ergebrusse der Anat u Entwicklung. Bd 16. 639. says that the renal artery is a trunk of a former mesonephric vessel utilised secondly as an inferior suprarenal, and finally, as a renal with the suprarenal as a branch. The photographs on pages 22 and 23 for instance are typicall of many cases, some of which help to support this claim, while others do not. Hochstetter F. 1906. Hertwigs Handbuch Vol 7. 116. and Hill E.C. 1905. John Hopkins Hosp Bull Vol 16 No 167 and Vol 17 No 181. suggest that in mammals other than man the renal artery is attained new from the Aorta, When the kidney reaches its permanent position. They say that in man a later vascularisation takes place from the aorta. But this is not in accordance with modern ideas of vessel growth, which maintains that new vessels do not sprout from an established trunk. Kolster R. 1901. Zeitschr Morph Anthropol Bd 4. 179 to 197. in order to explain renal arterial anomalies falls back on a theory of the utilisation of a late branch from some near by vessel.

From a close study of all the accessory vessels I have seen, and from consideration of embryological principles the theories of Brenner J.B. concerning the origin of the main and accessory renal vessels are easier to understand and seem nearer to the mark. (See Amer Journ Anat. 1915. 18. 179 to 200.) These theories can be summarised as follows according to him.

- (1) Anomalies of the renal artery depend on vessels present in the embryo before the aorta and its larger branches develope mesodermal coats. This is in 10 to 15 mm embryos. There are no late branches.
- (2) Certain anomalies are due to persistence of early renal blood supply as seen in pelvic kidneys for instance. See photograph on page 13 of this thesis.
- (3) A peri-aortic plexus with many roots from the aorta exists in many mammals, so affording opportunities for the change of position of the main aortic branch.
- (4) The renal artery is derived from this plexus or from such parts of it that exist just before the kidney gets its capsule.
- (5) The channel for the renal artery is selected MECHANICALLY from amongst many. (This is easier to believe than the theory of renal buds with some power of chemo or other type of taxis to draw vessels to them from the aorta.)

(6) When a channel which is mechanically convenient is occluded, non convenient channels are utilised, hence the anomalies. (One might add to this that sometimes more than one mechanically convenient channel is utilised at the same time, to give triple supply effects as in vessels a, b and c in the picture on page 47 of this thesis.)

A study of my own statistics and others lends support to this idea of a rich network around the aorta and deriving branches from it. Surely the network would be oriented to and richer on the left, owing to the richer blood supply required by the spleen, pancreas, stomach etc on that side. In view of this one would expect more accessory arteries on the left. The statistics bear this out. See those of Liedowtsch J.B. Arch f Klin Chir. lxxxix 1071. See also my own statistics on page 51A of this thesis. Grey's Anatomy. 28th Edition also confirm the fact that the majority of extra vessels are on the left side.

A method of dissection to reveal accessory vessels.

The kidneys lie behind many and important structures and are surrounded by fat, fascia, vessels, nerve plexuses, large veins and other structures. If they are simply grasped and cut off the aorta, large and small accessory vessels will be missed.

My method of study in every case has been what I call the Block Method. A complete block of tissue is dissected out containing both kidneys and suprarenals with their aortic connections untouched. In the block will also be the great veins, nerves, and parts of adjacent muscles such as the diaphragm. Such a block after taking out, looks like that in the photograph on page one. Dissection is seen to have been started in this one, and already a low accessory artery (ra) is seen in the process of being uncovered.

Before taking such a block out I start by carefully dissecting out the contents of the thorax, keeping watch for thoracic connections with the renal vascular system. The diaphragm and the liver are then separated, and the liver is taken out, leaving behind the right supra renal gland and still keeping a careful watch for vessels. Then the descending colon is cut very low and all the bowels dissected out with pancreas and duodenum, to leave behind the aortal renal block, which is cut out by incisions as low as the pelvic level and as high as the aortic arch. The block when taken out is then taken out and dissected carefully, feeling for delicate vessels with the bare fingers. The final result of work that sometimes takes some hours is shown on Page Two.

Observations on the structure of the Vessels.

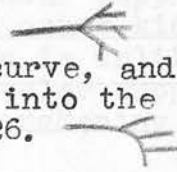
Sections of vessels can be cut and studied to see what differences there are, if any, between main and accessory vessels. See photographs on pages 38, 39, 41, 42, 44, 46, 48, and 50. These kinds of stained sections thrown from an enlarger to photographic paper give an idea of comparative coat thickness of the different vessels. Sections can be stained by silver methods to show the plentiful supply of nerves in the coats as in the microphotographs on pages five and six.

In a study of very many dissections I could not find any marked differences between main and accessory vessels from either nervous supply or musculo-elastic coat structure point of view. Some of the accessory vessels have occasionally thin flabby vein like walls. (page 35.)

The pattern of the branching of the renal main vessels do not by a long way always confirm to the simple division as depicted by Max Brodel in many books.

(A Max Brodel renal artery is seen in Fig 209 Page 533. Cunhnm Manual Pract Anat. 6th Edit. 1914.)

More often than the Brodel even branching we see the main vessel taking a sweeping curve, and sending out a series of parallel branches into the kidney as in photographs on pages 29 and 26.



Some types of renal accessories, usually the thin coated vein like ones are just smooth tubes and never have any branches. Such a vessel is seen in the photographs on pages 30 and 35.

The orifices of accessory arteries are usually poor affairs and prone to distortion and blocking, and they are set at a different angle.

Observations on the pathology of the vessels.

The accessory arteries are very prone to atherosclerotic changes, especially when they are small and poorly developed. In spite of their partial obstruction in many instances, and the comparative ischaemic state of the kidney area served, in no instance did I find clinically in life a high blood pressure.

Calcium deposition in the vessel coats was not so common as expected, in fact generally rare in my cases. This was surprising considering the very common grossly calcified state of the aorta. (See example in photograph on page 17.)

Another part in the pathology of the kidney which according to many authors is played by accessory vessels, is the part of producing hydronephrosis. The usual description is that an aberrant renal artery kinks the pelvi-ureteric junction to eventually cause hydronephrosis. I have examined very many such cases of low accessory vessels, many of which are pictured in this thesis, and I cannot say that they took part in causing hydronephrosis in any instance.

Often when a low accessory vessel is seen on one side only, a greatly enlarged pelvis of the ureter is seen to be bilateral. The number of times the enlargement is seen without an obstructing vessel, and without any obstruction elsewhere gives one the opinion that this enlarged state of the pelvis is a congenital atonic condition like the condition seen in megalocolon. (Hirschsprung's Disease.) Some prolapse of the kidney may get the enlarged pelvis to overhang a low artery occasionally with obstruction. The innocence of the renal accessories in this condition is also confirmed by

Boyd W. Surg Path. 5th Edit. 1942. 355.

White H.P.W. Brit Jour Surg. 1925. 13. 247.

Hinman F. J.A. Med. Assoc. 1924. 82. 607.

Hinman F. Arch Surg. 1926. 12. 830.

In the case of infarction of the kidney the cortical blood vessels play a considerable part. There is a capsular capillary circulation present in the kidney, with offshoots to fat, fascia, and to join muscular and lumbar anastomotic branches. Because of this capsular supply the infarct does not reach the surface, the outer kidney tissue being nourished by this supply. This is unlike the spleen for instance, where there is no capsular supply, and the infarct reaches the surface with roughening and adhesions.

These observations show that much help can be gained in understanding renal pathology by an attempt at classifying and examining all the different vessels that take part in nourishing the kidneys.

Accurate classification helps to know and study the reserve and collateralisation power of the kidney.

Such study also helps as a guide for the surgeon who has to operate on or remove the kidney.

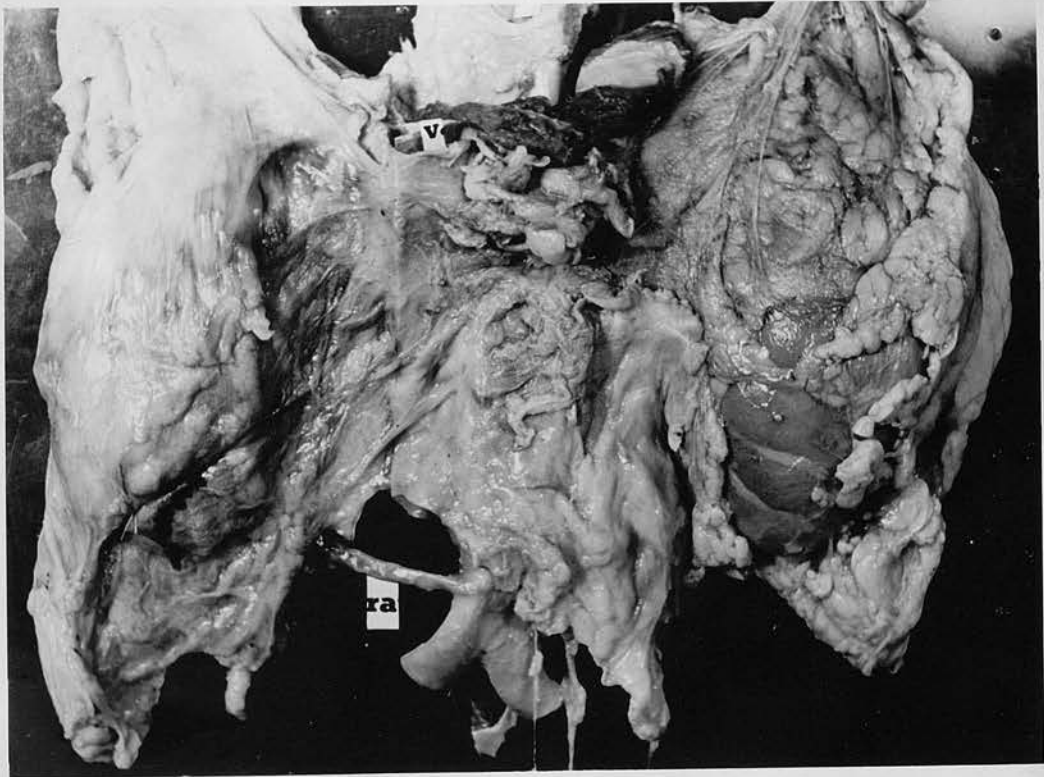
In my studies my statistics have helped to give me the opinion that rather than being the normal state, the kidney with only one artery is very much in the minority.

Hypertensive changes in a human kidney where one vessel is partly obstructed are not common, and it seems that if such changes are established there is fair chance of ameliorisation from collateralisation.

Abnormal renal arteries have been quoted as being the cause of high blood pressure, but in my series of many cases I have not come across this.

Summary of observations on Renal Vessels.

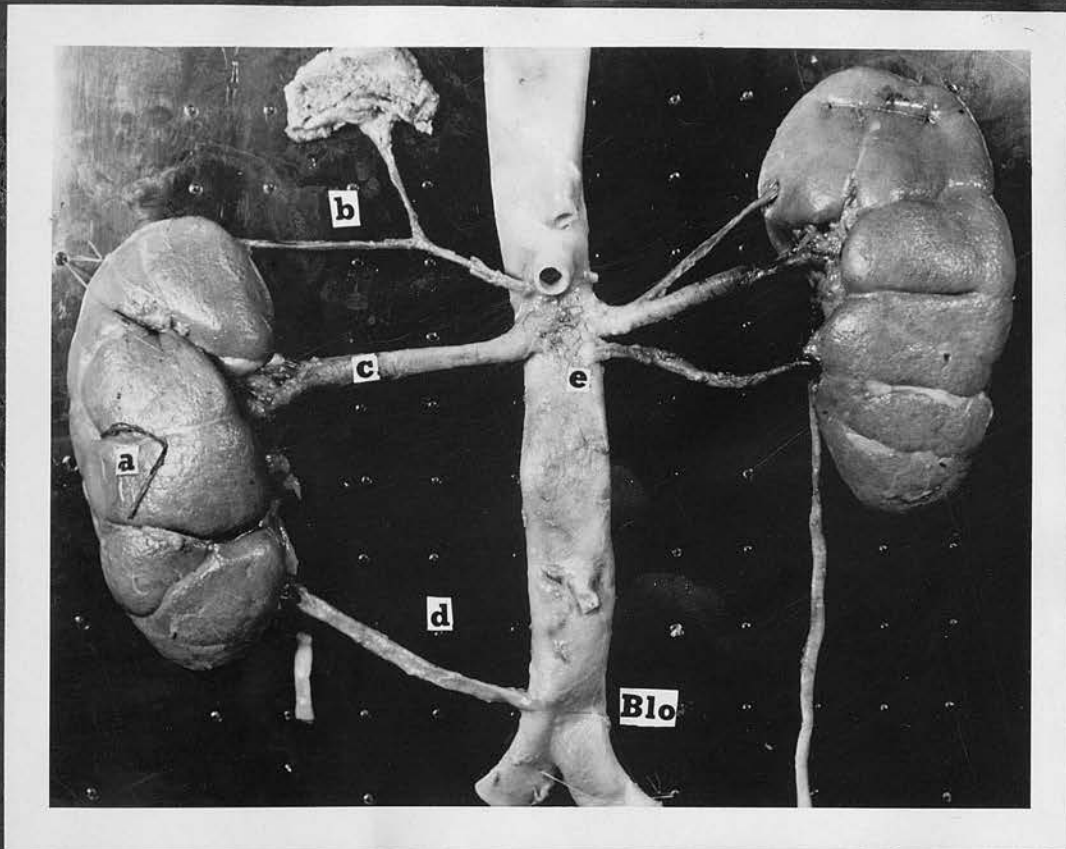
- (1) The importance of the renal blood supply is discussed. The work of Goldblatt and others is reviewed and analysed.
- (2-) The embryology of the renal vessels is discussed and findings compared.
- (3) A method of dissection of the renal vessels is described.
- (4) The anatomical structure of the renal vessels is discussed, including relations with the suprarenal.
- (5-) Points in the pathology of the renal vessels are discussed, in their relation to hypertension, calcification, infarction. and hydronephrosis.
- (6) Statistics are shown to show that accessory vessels are present in over 90% of cases.
- (7) Photographs of different types are shown.
- (8) The main findings are
 - The presence of accessories in 90%.
 - Lack of hypertension in spite of occlusion of arteries.
 - Accessory vessels seldom if at all cause hydronephrosis.
 - A close nervous and vascular connection between kidney and suprarenal gland.



PHOTOGRAPH OF BLOCK OF TISSUE DISSECTED OUT (PARTIALLY)
FOR STUDY OF RENAL CIRCULATION.

ra = shows an accessory artery from aortic bifurcation
to lower renal pole appearing.

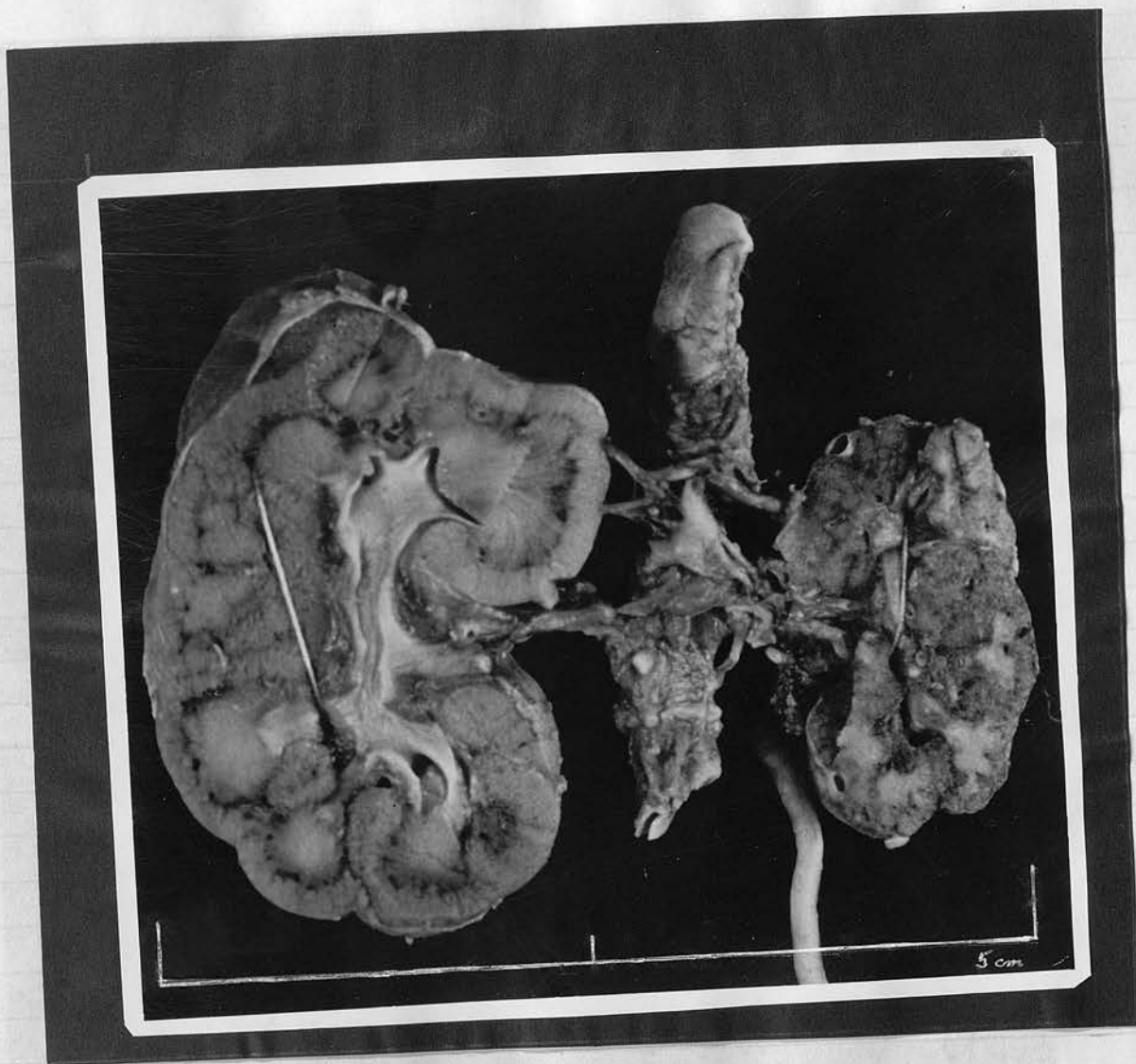
V = opening of vena cava.



PHOTOGRAPH SHOWING BLOCK IN PREVIOUS PHOTO FULLY DISSECTED OUT.

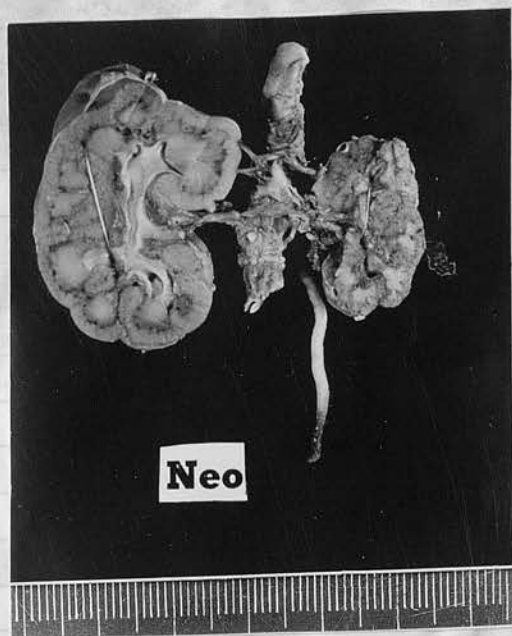
- d = accessory artery (caudal. polar.) coming from bifurcation area aorta.
- c = Main renal artery.
- e = accessory below main left renal artery which could easily be missed.
- b = accessory (cephalic. polar.) giving branch to suprarenal.
- a = small artery leaving kidney for fascia to anastomose with other small arteries (cortical fascial type).

Ureter unaffected by d.

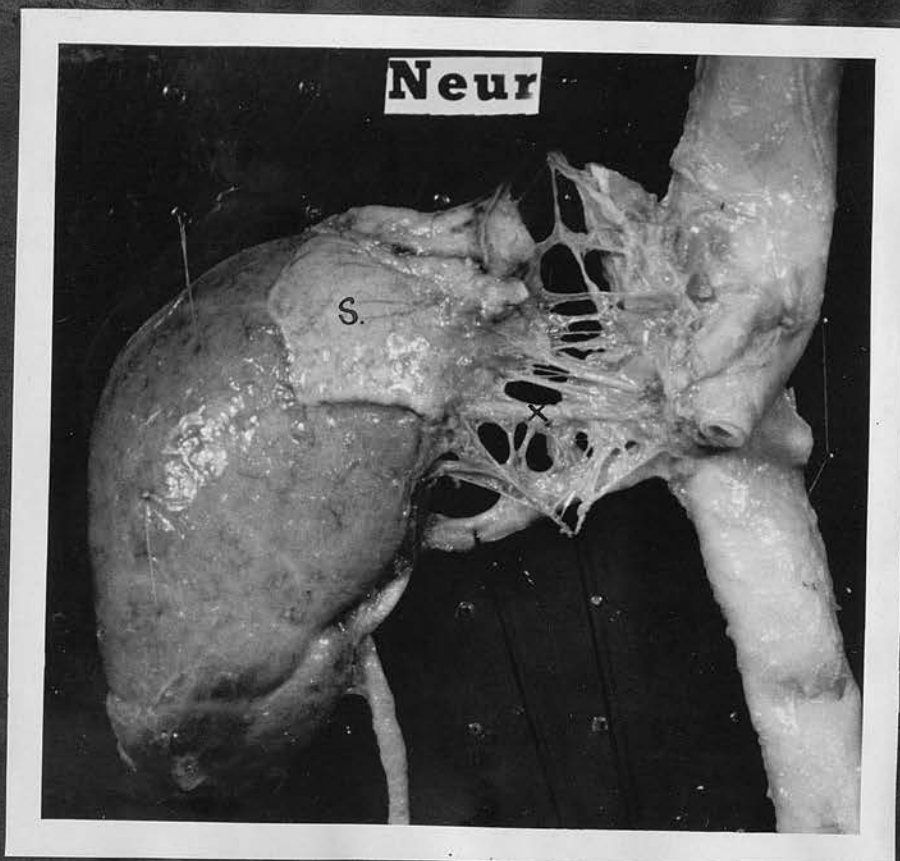


Photograph of foetal
Kidneys to show →
accessory arteries.
above same enlarged.

upper or cephalic
types of accessories
seen right and left.



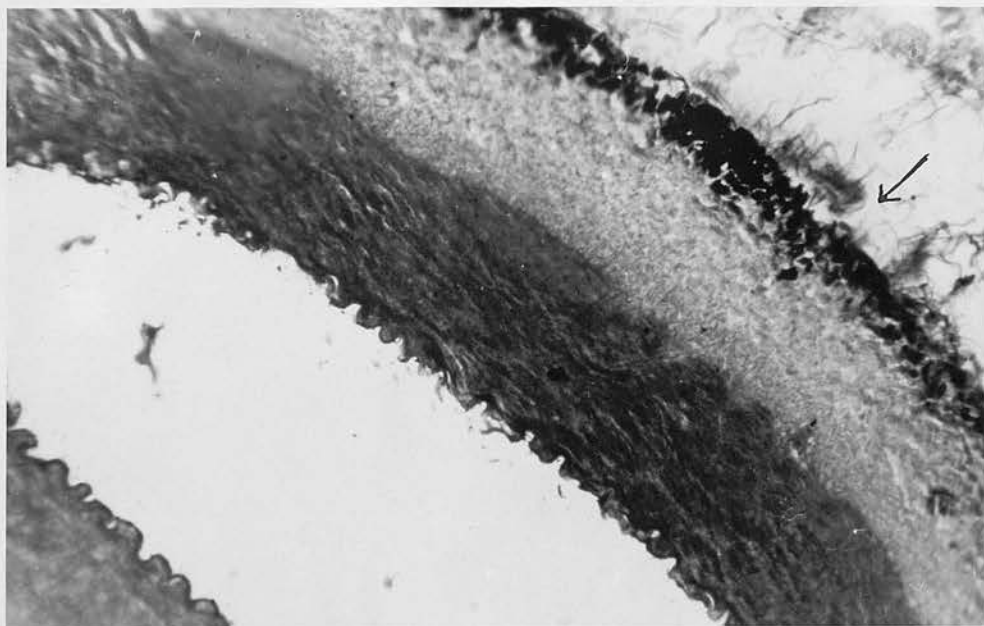
← normal size.



PHOTOGRAPH SHOWING association of accessory artery X.
with nerve supply.

Illustrating also difficulty of dissection
and ease with which accessories can be
missed.

S = Suprarenal gland.



MICRO PHOTO SHOWING GOOD NERVE SUPPLY OF AN ACCESSORY.

also shows well the coats of the artery.

Nerves shown black in this stain opposite arrow.

Details of Stain

Fix in Abs Alc with 1% ammonium hydroxide 48 hours.

Rinse distilled water

Refix in Pyridine 24 hours.

Wash in distilled water 24 hours.

Leave in 2% aqueous solution of Silver nitrate in dark at 35°C for 3 days.

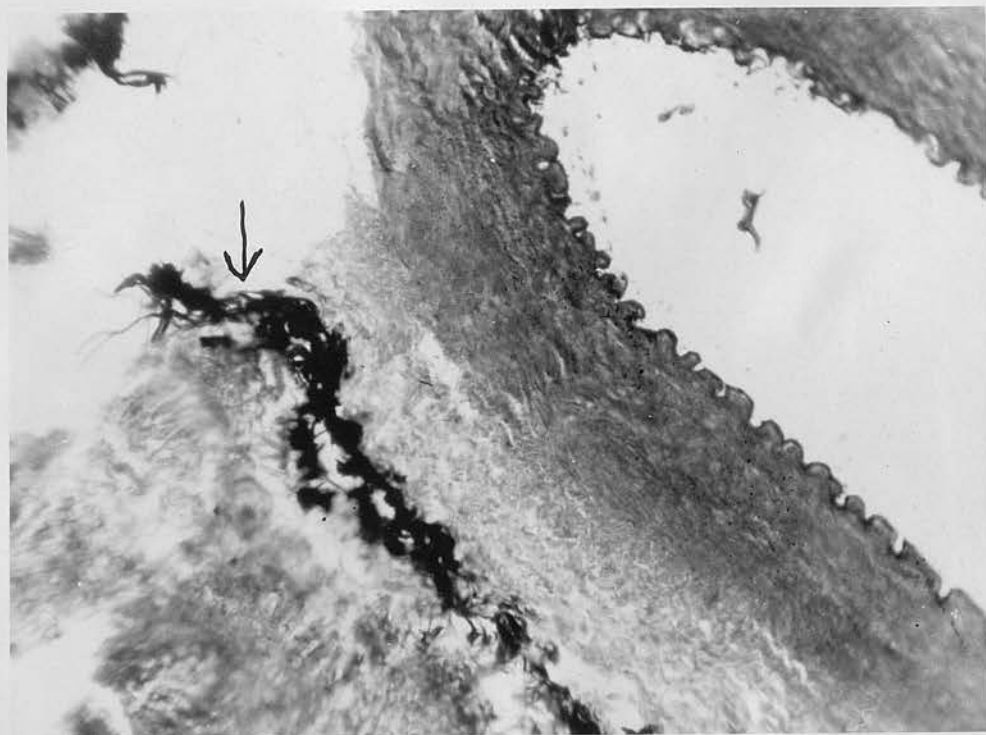
Rinse in distilled water.

Reduce in 4 gm of Pyrogallol in 100cc of 5% Formal in aqua dest for 24 hours.

Wash in distilled water. Dehydrate. Embed in Paraffin.

With this stain tissues are varying shades of brown.

Axons of both myelinated and unmyelinated nerve fibres black.



MICRO PHOTO OF ANOTHER ACCESSORY ARTERY.
SAME STAIN

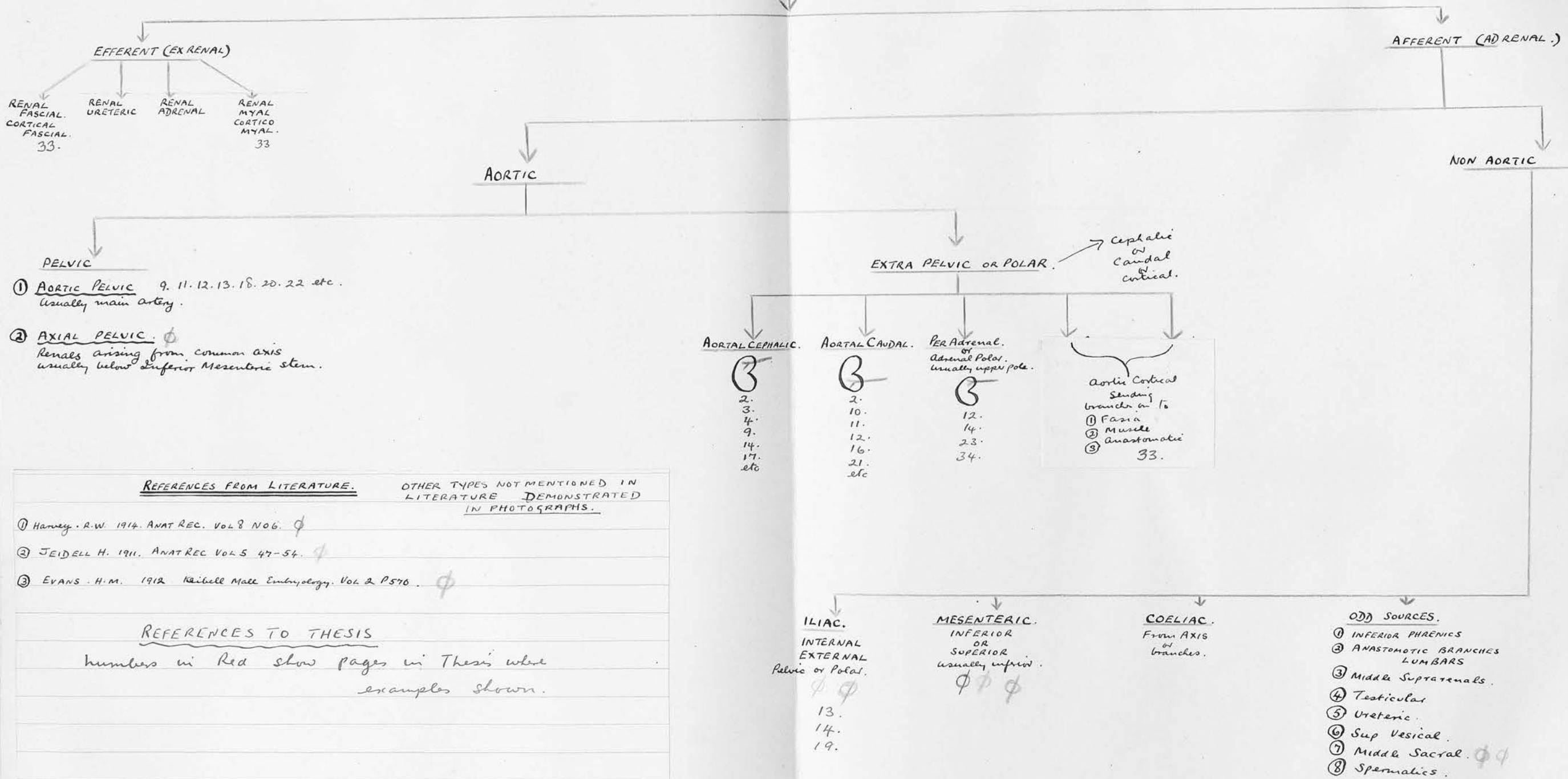
Arrow shows nerve fibres.



It is often the case when Aorta badly calcified — renal vessels do not become calcified.

SCHEME OF CLASSIFICATION
OF
RENAL VESSELS.

RENAL ARTERIAL SUPPLY AND SYSTEM.



REFERENCES FROM LITERATURE.

OTHER TYPES NOT MENTIONED IN LITERATURE DEMONSTRATED IN PHOTOGRAPHS.

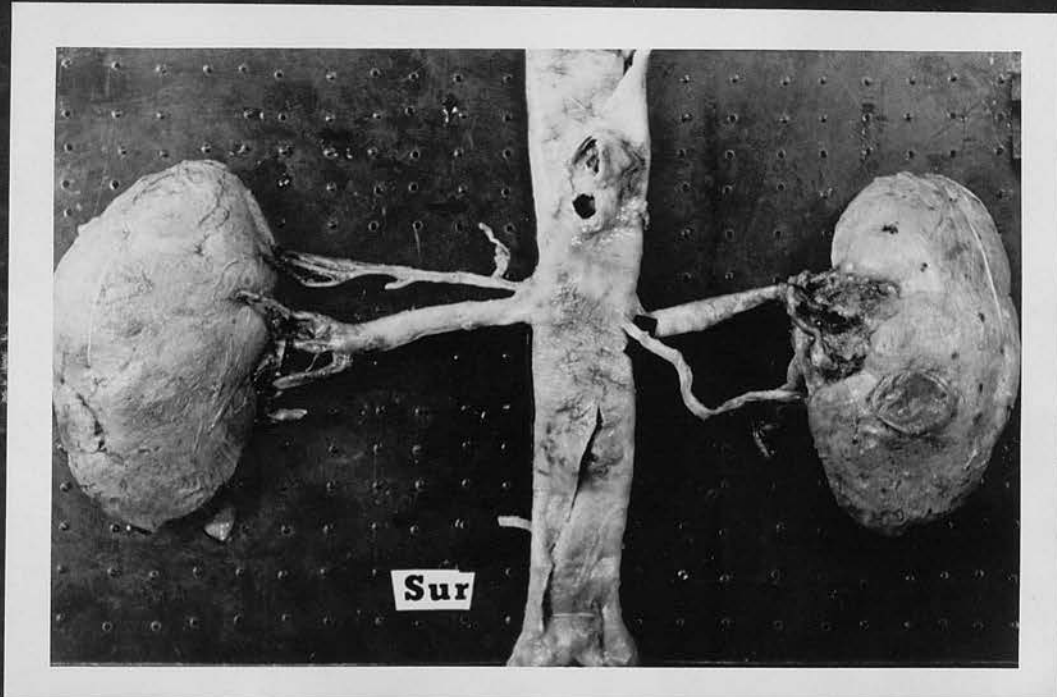
1 Harvey. R.W. 1914. ANAT REC. Vol 8 NO 6. ϕ

2 JEIDELL H. 1911. ANAT REC Vol 5 47-54. ϕ

3 EVANS. H.M. 1912. Keithell Male Embryology. Vol 2 P576. ϕ

REFERENCES TO THESIS

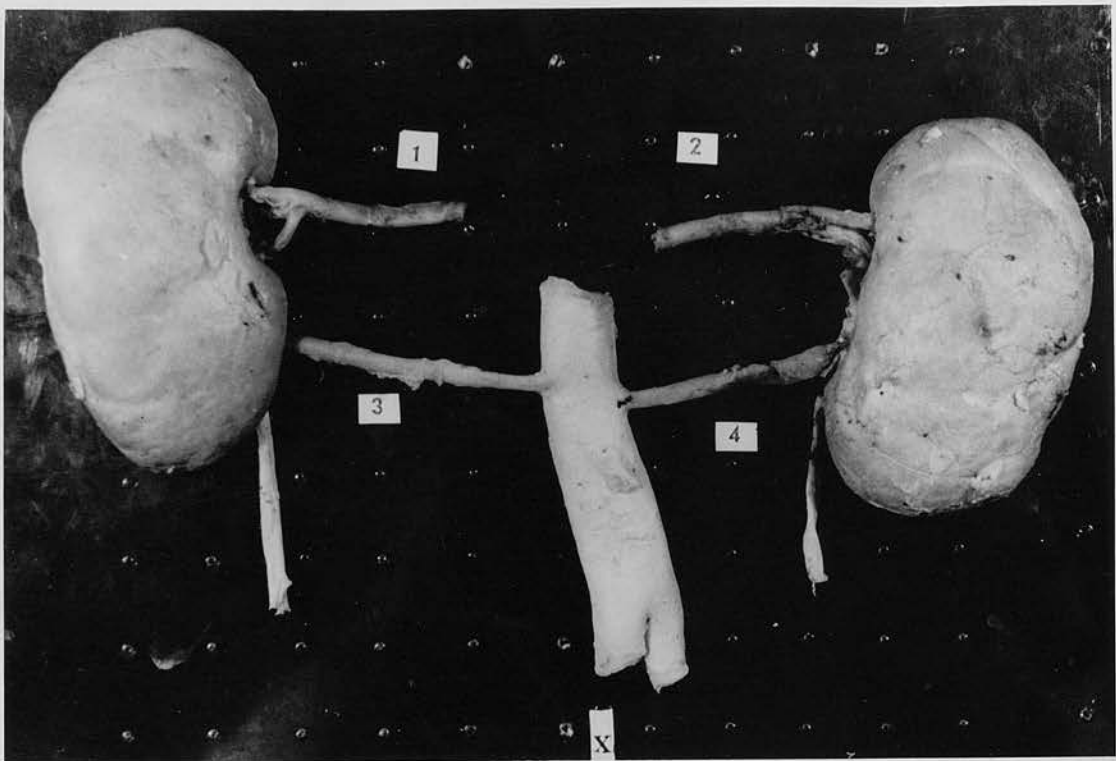
numbers in Red show pages in Thesis where examples shown.



TYPE OF ACCESSORY VESSELS.

Bilateral from main renal artery base.

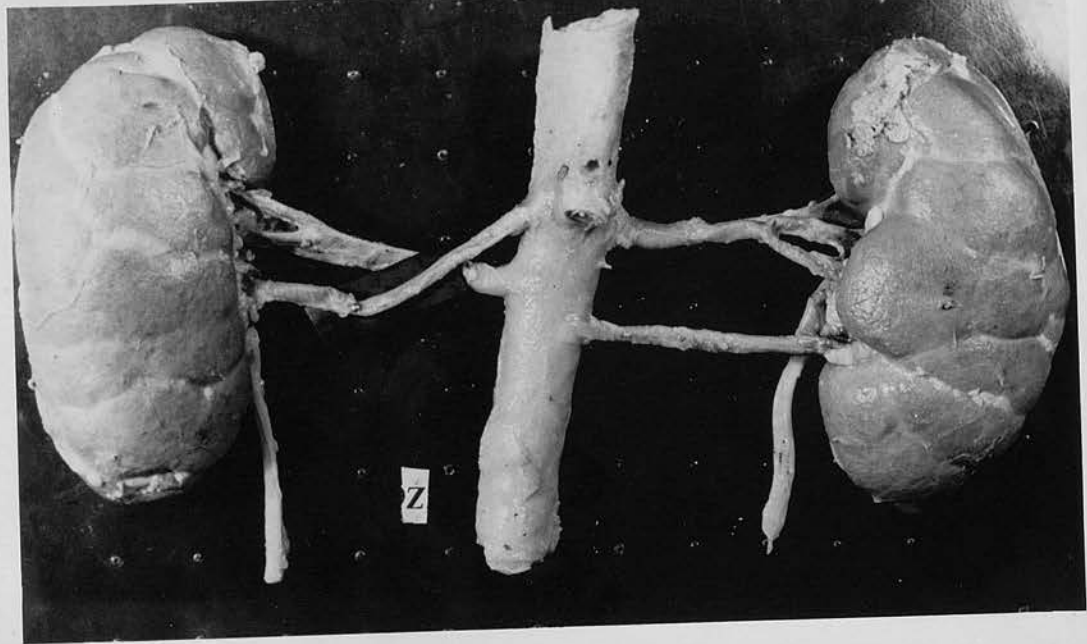
Delicate structures liable to disease changes and occlusion.



Types of Renal accessones. (3 and 4 in photo)

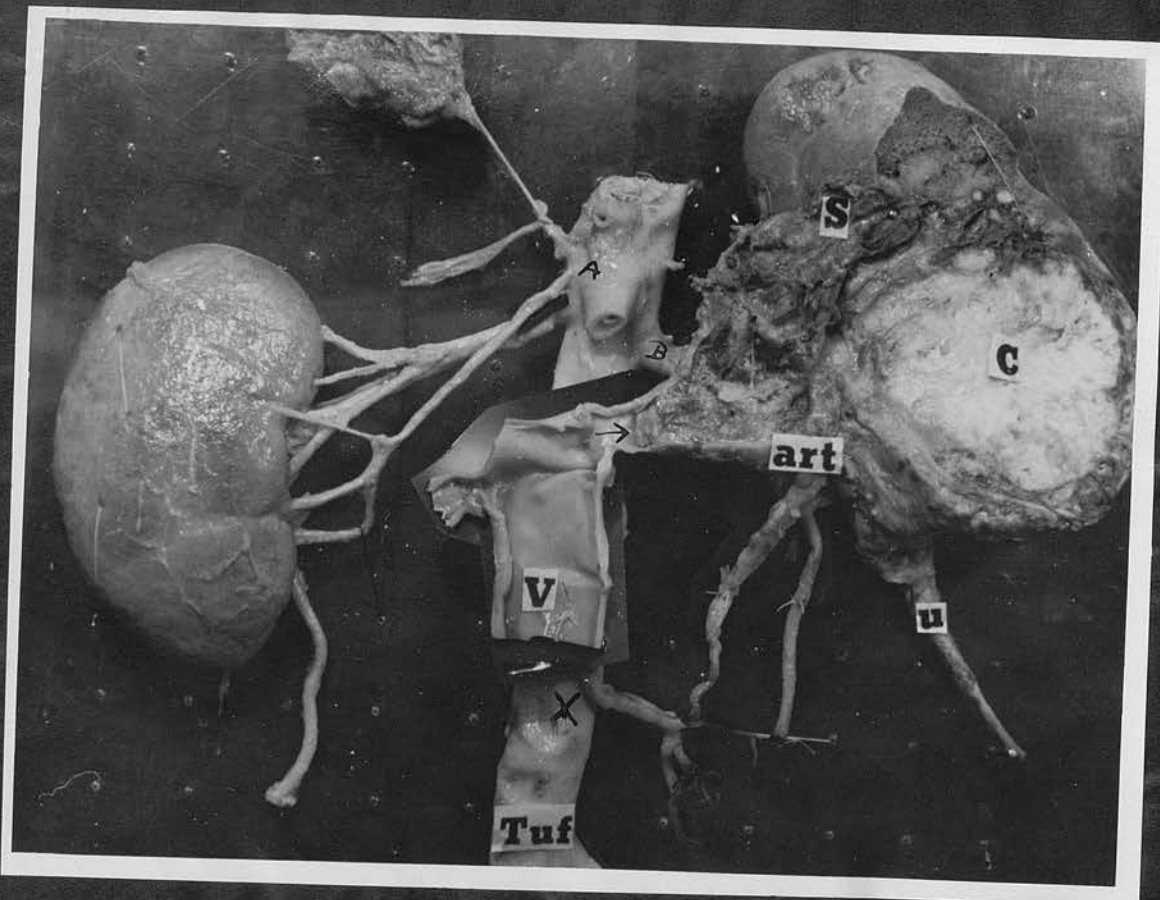
Robust accessones bilateral going towards end of pelvic area rather than lower pole.

Main Renals and Aorta where they came from cut off for study.



Types of Renal accessories.

On right crossed accessories which is not uncommon.
 On left accessory low to lower pole.
 No interference with ureters.



Accessory Renal arteries displaced by "hypernephroma".

C = new growth.

→ points to advancing edge of cancer cells in renal vein.
There was a sharp edge here to the advancing mass.

V = Vena cava.

u = displaced ureter.

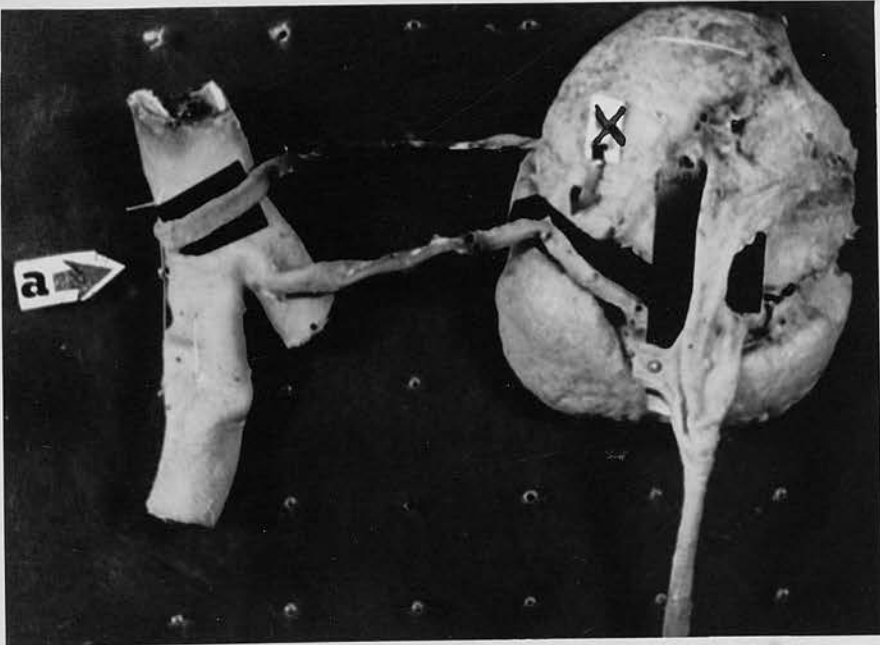
ART = displaced two accessory arteries.

B = main left renal artery.

S = Suprarenal.

A = Right crossed accessory artery.

X = origin? of one of the left accessories.



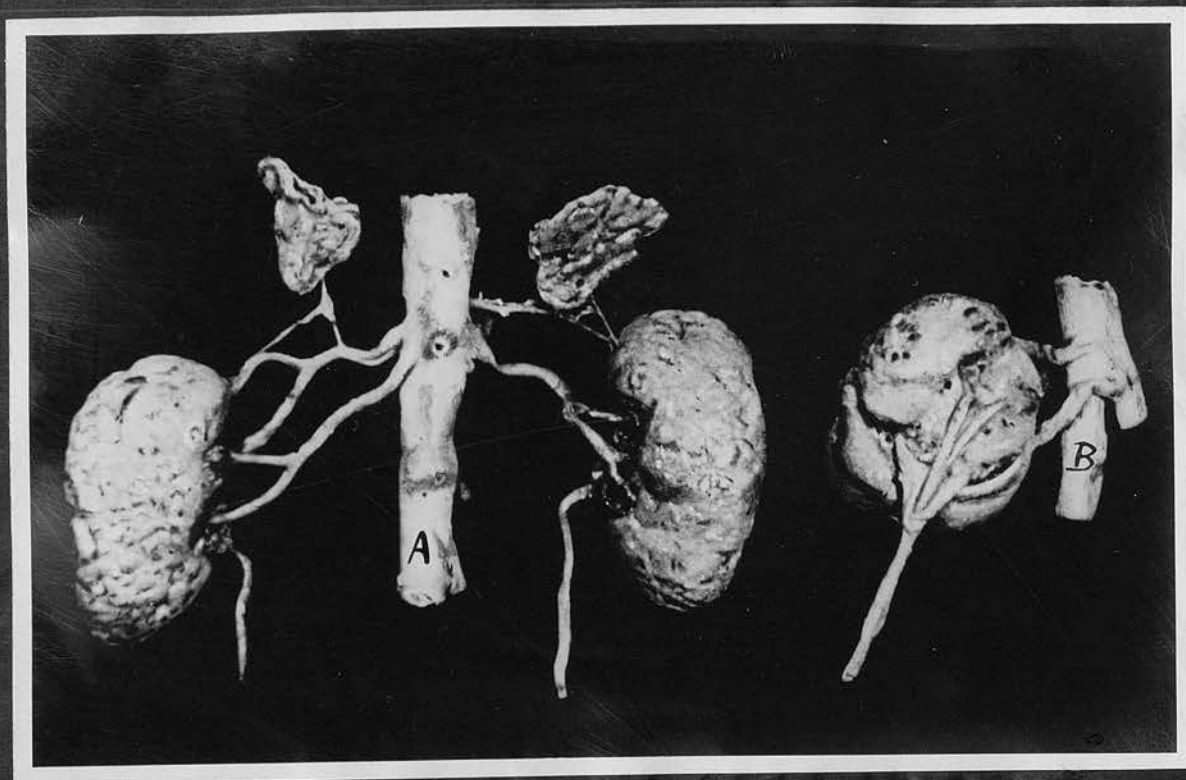
The BLOOD SUPPLY OF ABNORMAL KIDNEY.
LEFT PELVIC KIDNEY.

Arrow A = points to artery supplying this kidney coming round from back of Aorta.

Arrow X = points to vessel coming to supply kidney from Common iliac.

Notice the Pelvis and ureter are placed entirely anteriorly.

No symptoms caused.



A

B

Right pelvic Kidney.

Two arteries supply from lower aorta.

Bilateral accessories.

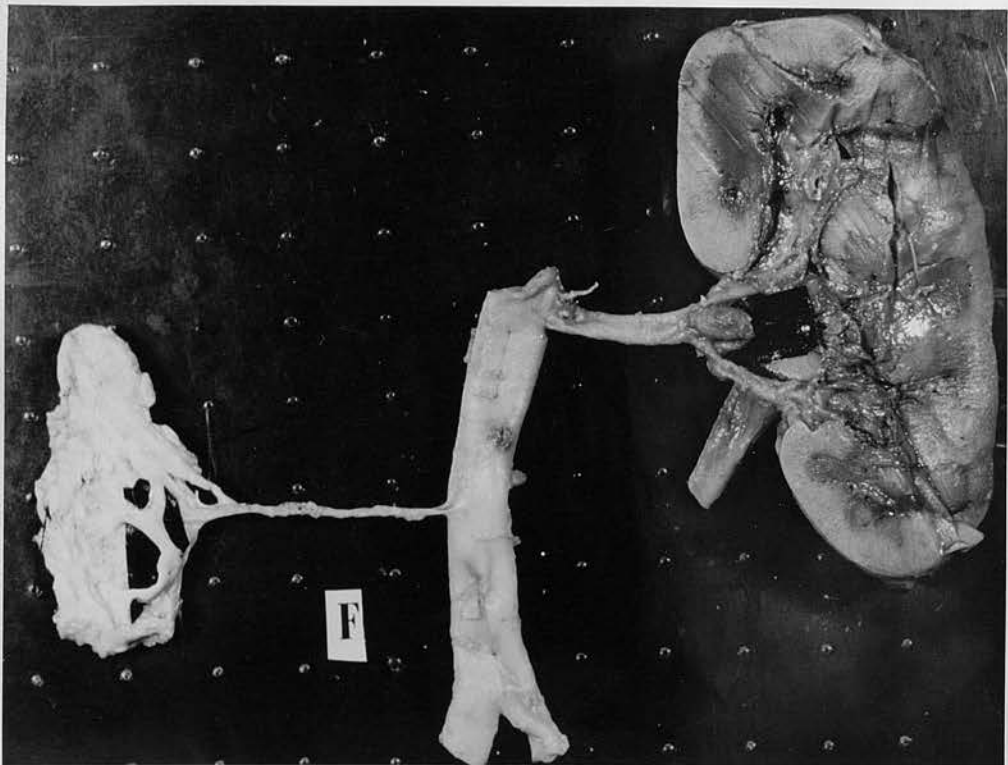
Well developed on right

with association with suprarenal.

Poor development on left

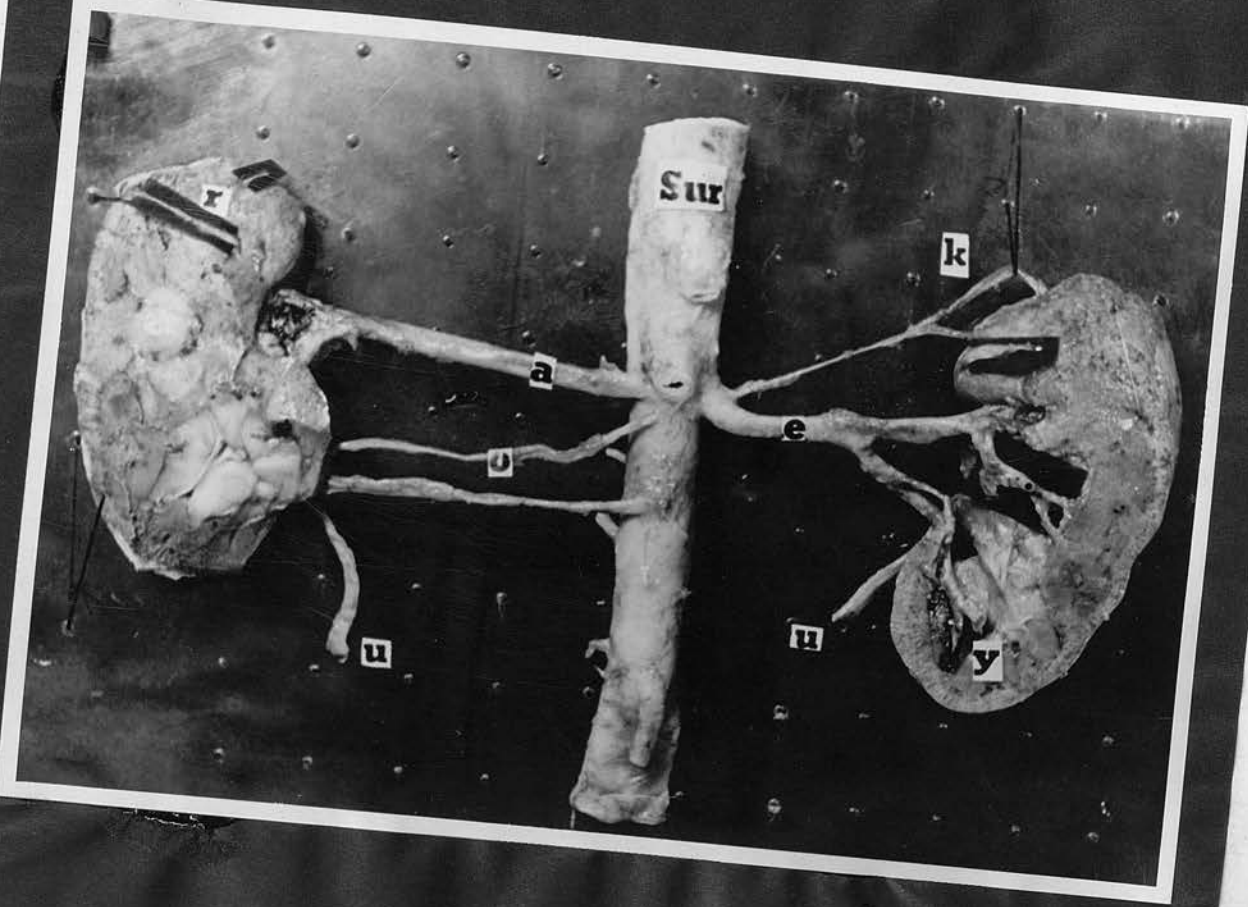
also with association with suprarenal.

Aortal pelvic and aortal cephalic types.



Supply of Rudimentary Kidney. (Right.)

note frail vessel stem but good sized ramifications.
also arising low down as compared with left.



Bilateral Renal accessory arteries.

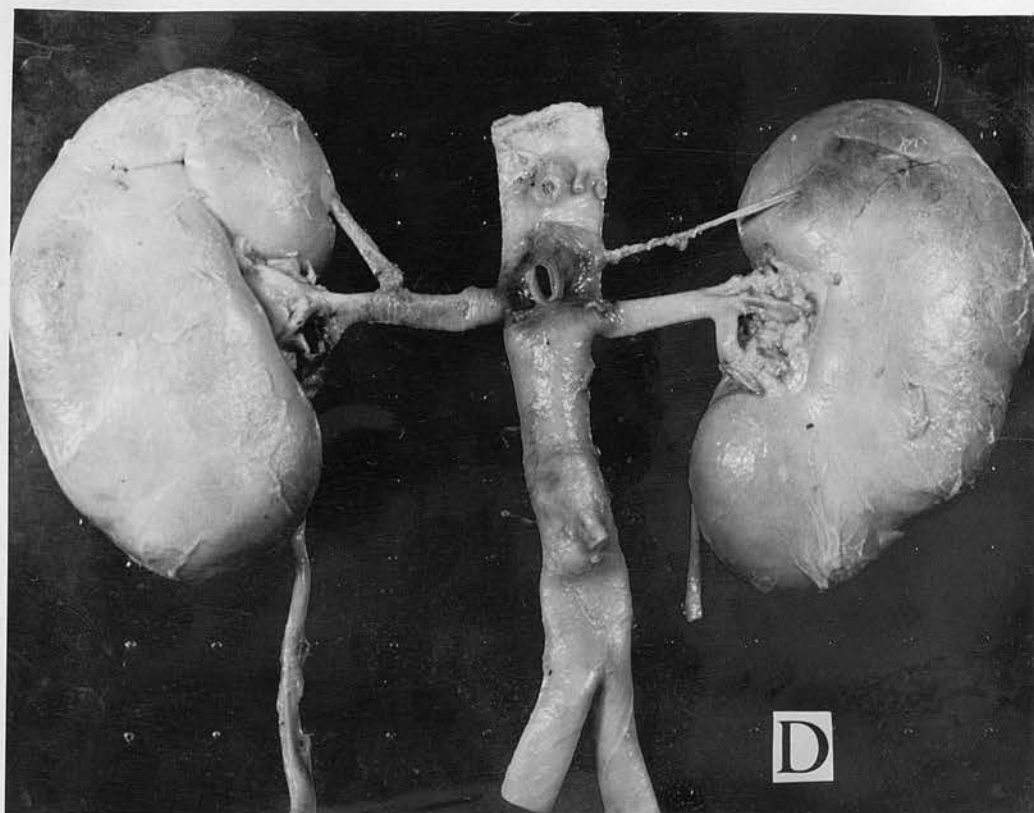
Note triple supply on right.

a = main branch.

O and artery underneath accessories.

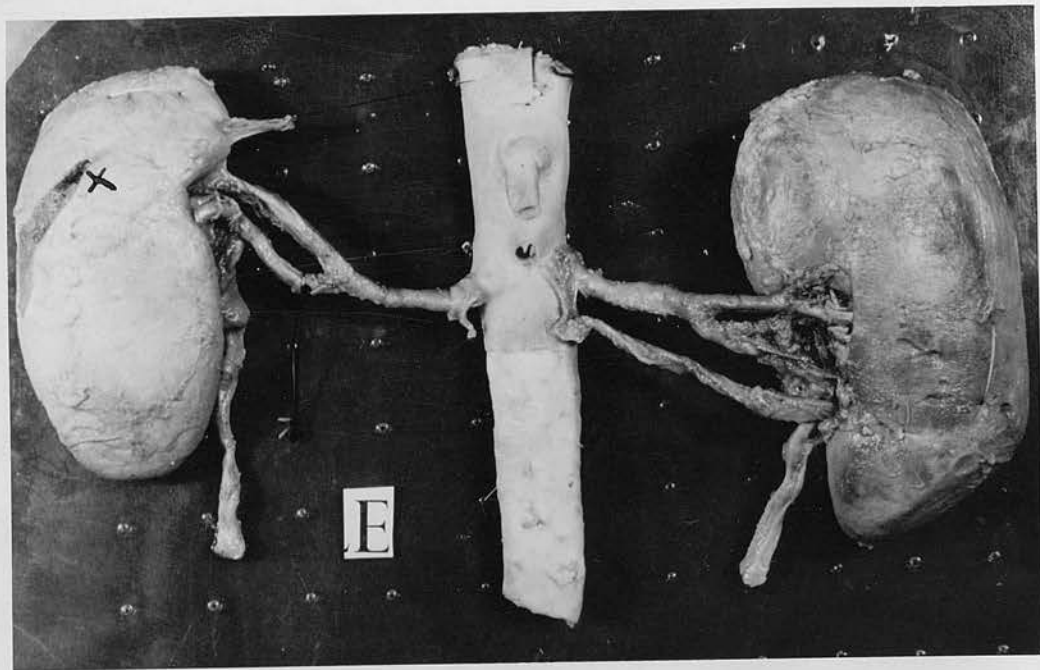
On left frail cephalic type of accessory liable to easy occlusion.

r = on upper pole right Kidneys show cortico fascial branch going out to upper fat and fascia.



Type of Renal accessory artery.

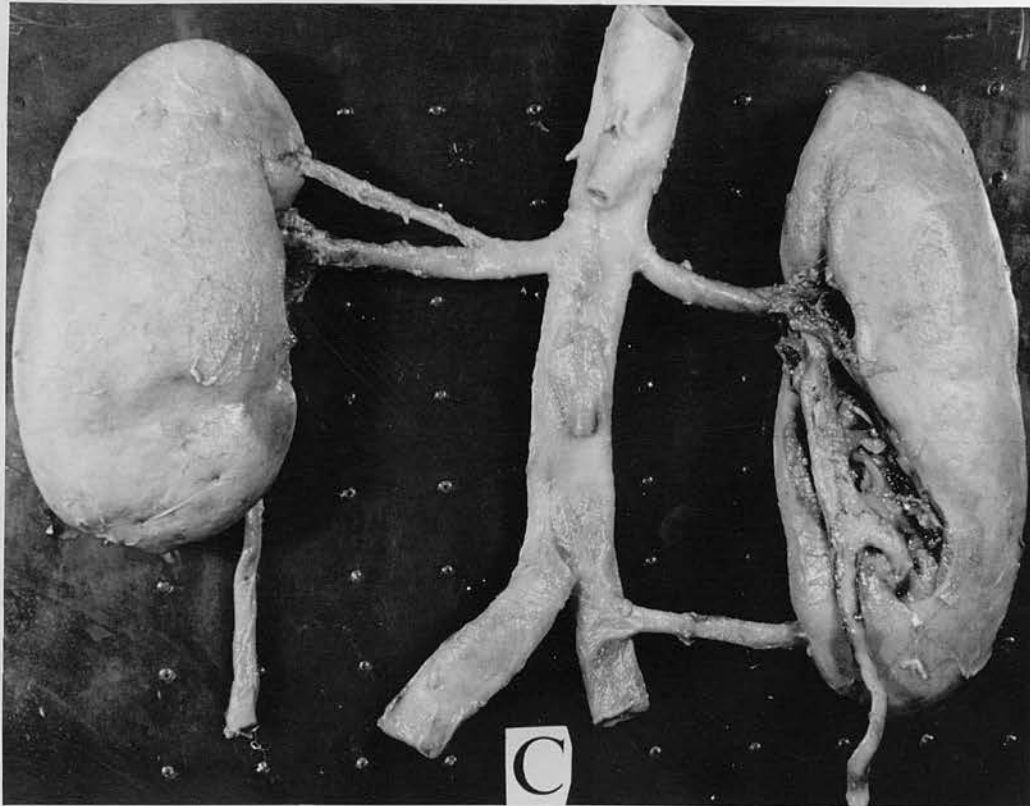
Small poorly built aortalcephalic type of accessory on left side.



Type of Renal accessory.

On left side low type of accessory going down to lower pelvis. Ureter showed no change.

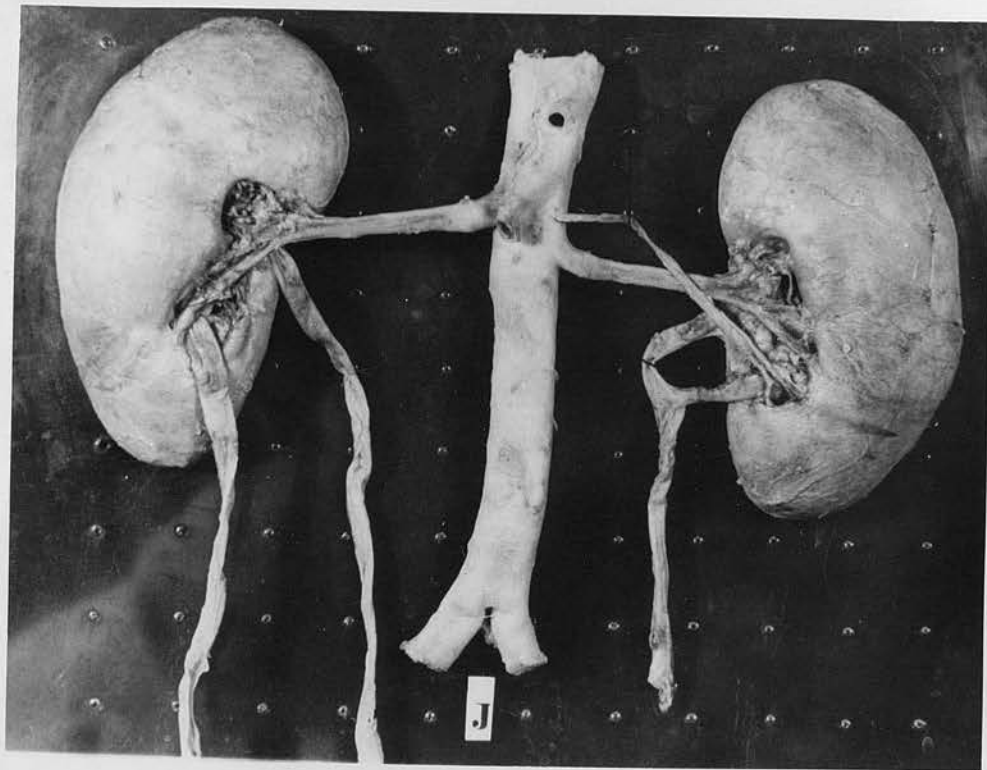
On right at X torn cortico fascial type of vessel.



TYPE OF ACCESSORY ARTERY.

Left sided ilio - caudal (polar) type.

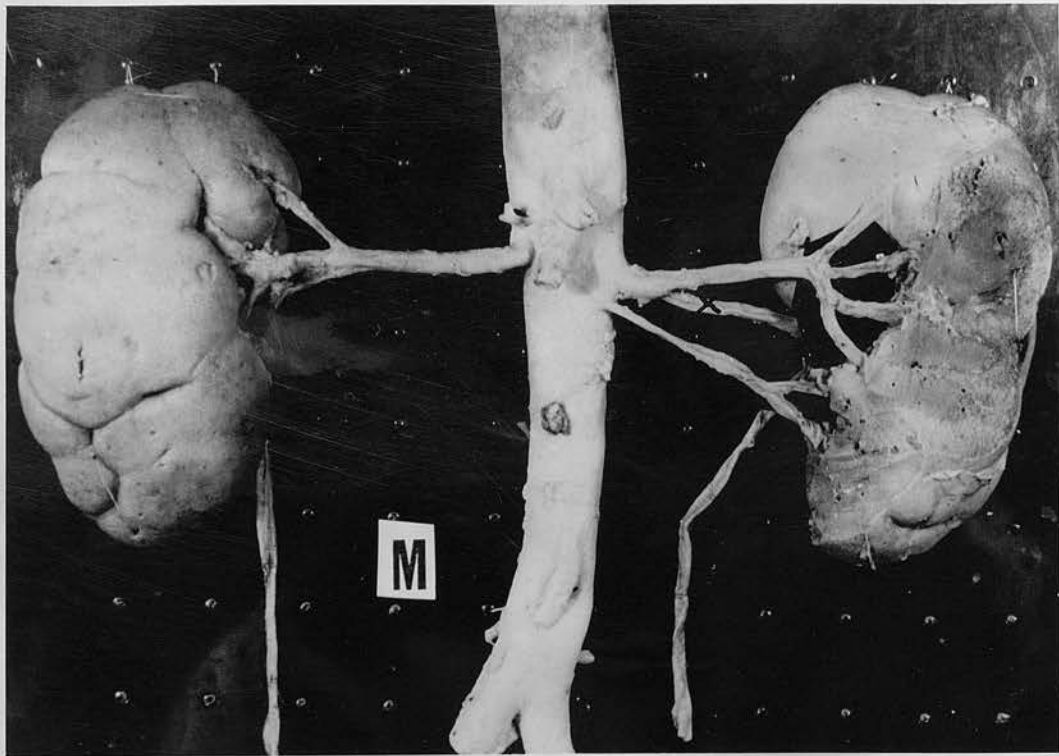
Note forward pelvis (non rotation of kidney)



TYPE OF ACCESSORY ARTERY.

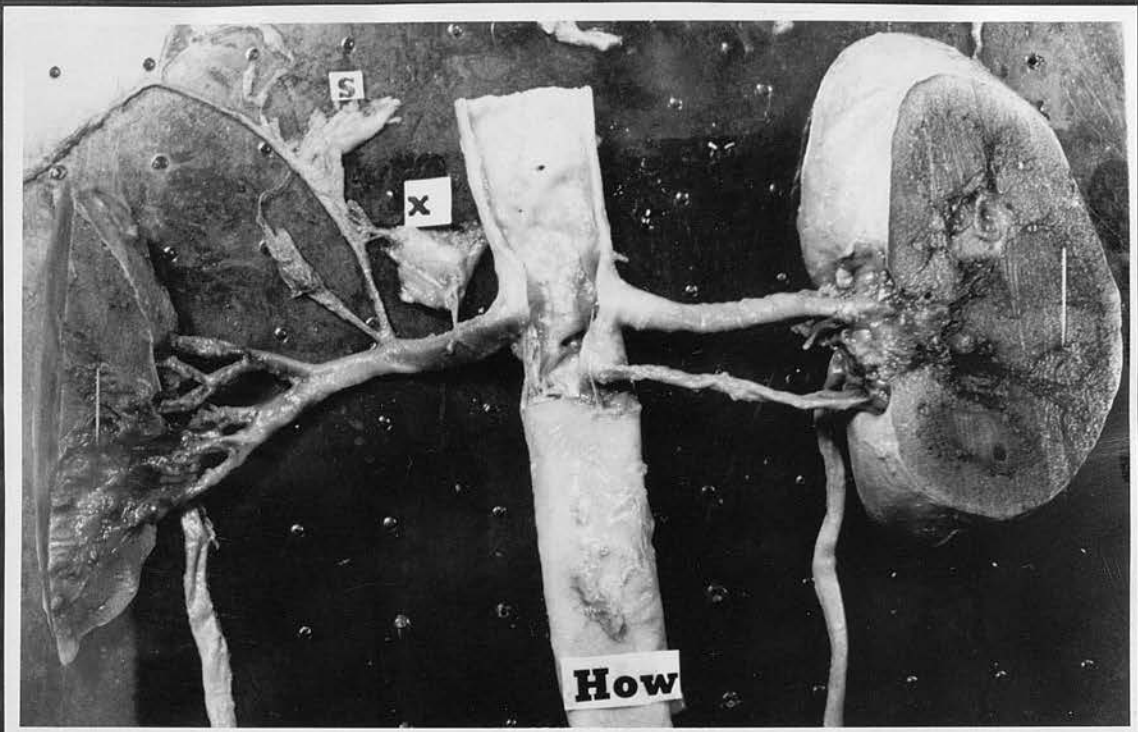
Left crossed type of aortal pelvic accessory.

Note on left bifid ureter.
on right Two ureters.



Type of accessory artery.

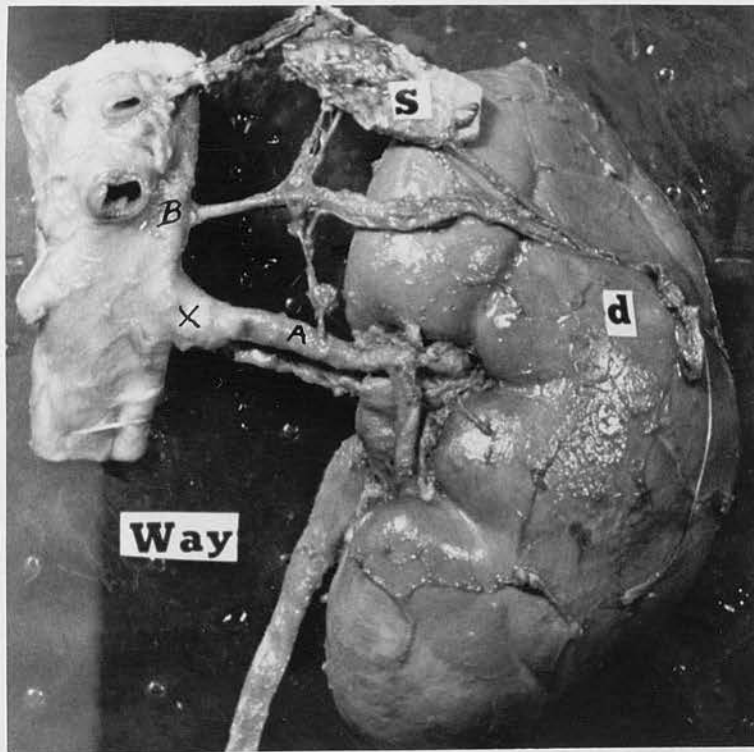
Left aortal caudal type. no interference with ureter.
 At back of main renal X another aortal pelvic
 accessory is seen.



Type of Renal accessory vessel.

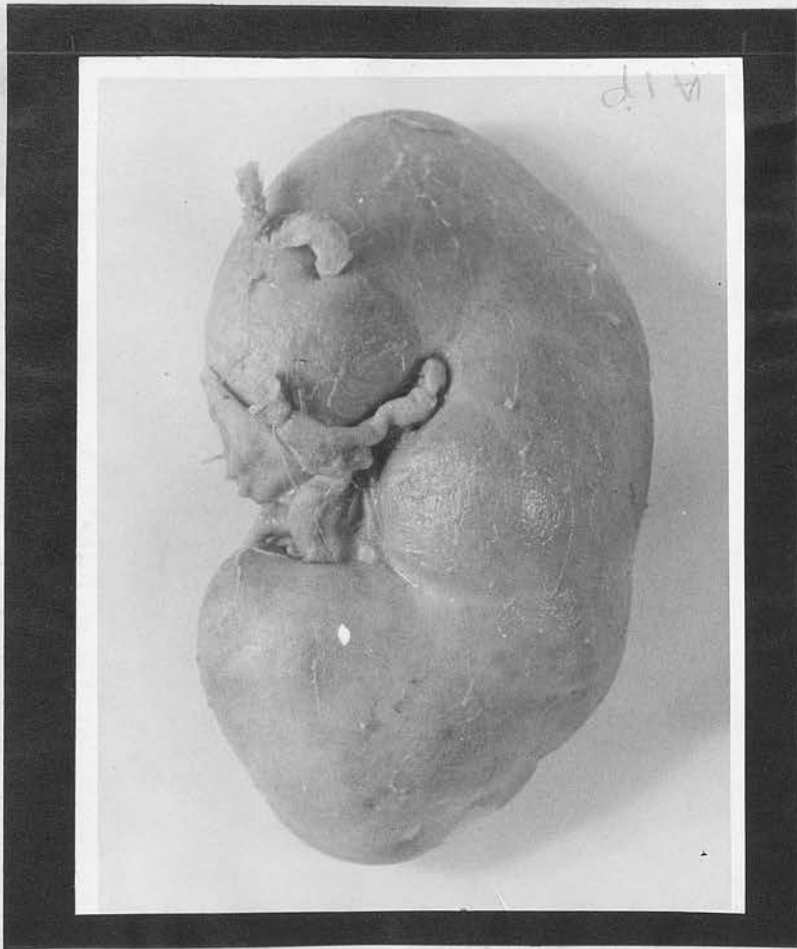
Left sided accessory branch going to lower end of pelvis.
no interference with ureter.

On the right shows blood supply to ganglion X
and suprarenal S.



Type of Renal Accessory Vessel.

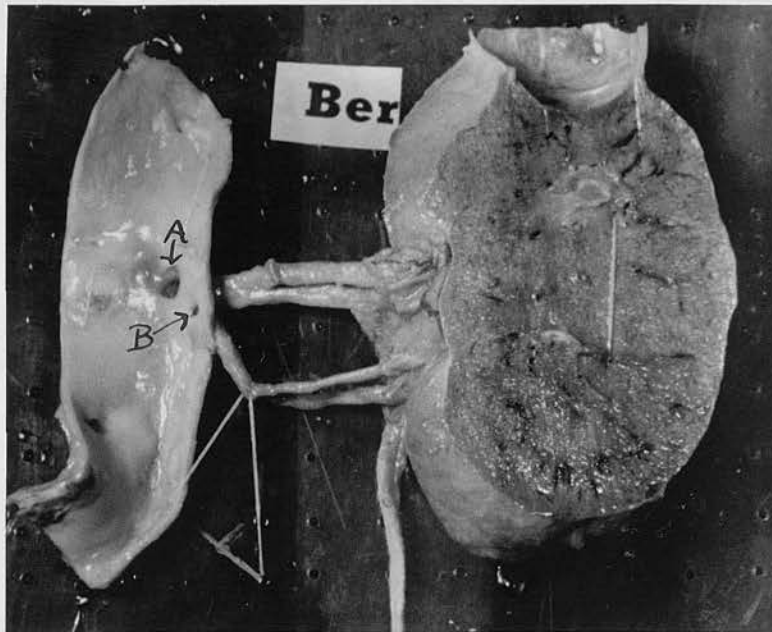
Here at X The main Renal vessel splits at origin into two parallel branches which beside the vessel but has not affected it. From it at A a branch goes up supplying a small ganglion and then joins the accessory B which sends branches to the suprarenal S and then proceeds to dip into the cortex in D area and send on a fascial branch. There is communication also between the d area and the suprarenal gland. In most cases and I think all the suprarenal and kidney supply are from the nervous and vascular point of view in close cooperation.



Aortal cephalic
type of accessory.

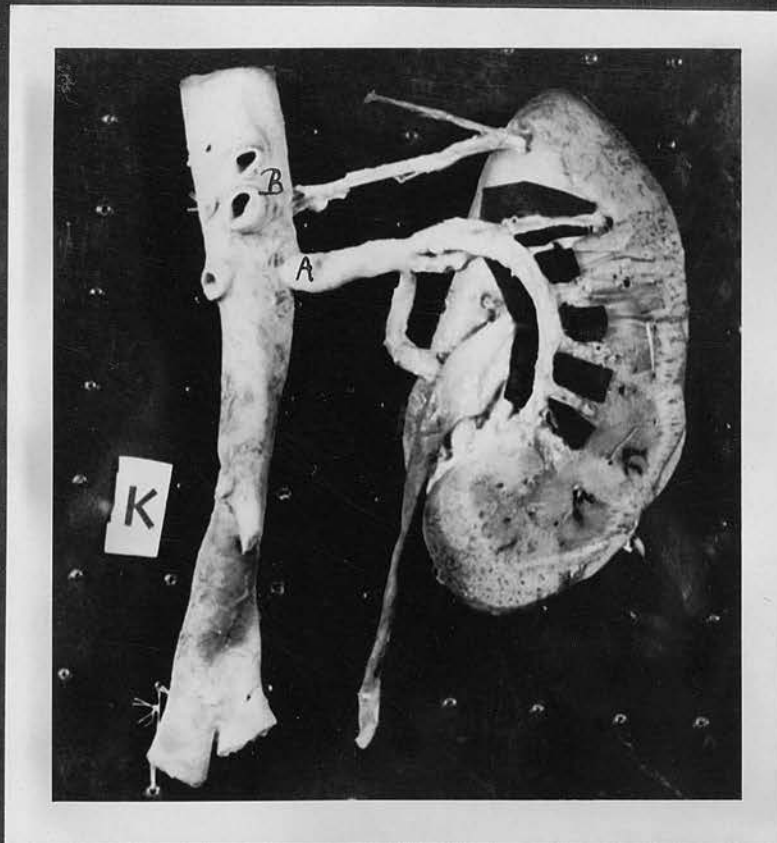


Radiograph of
same injected to
show area of
supply of accessory
and part of the
main renal.



Type of Renal Accessory Artery.

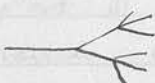
- A shows opening of main bifurcated renal artery.
 B shows opening of accessory renal artery. Aortal Pelvic type.
 Note the small opening for the comparatively large vessel.
 Many of these vessels are half obstructed at their openings with diminished blood supply and ischaemia of area supplied — but with no increased blood pressure in life.



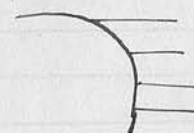
Type of Renal Accessory artery.

A shows main Renal artery.

Very many of these do not branch in the way as shown in conventional drawings such that of Max Brodel (Cunningham's Manual Pract. Anat. 6th Edit. Vol 1. Page 553 Fig 209)

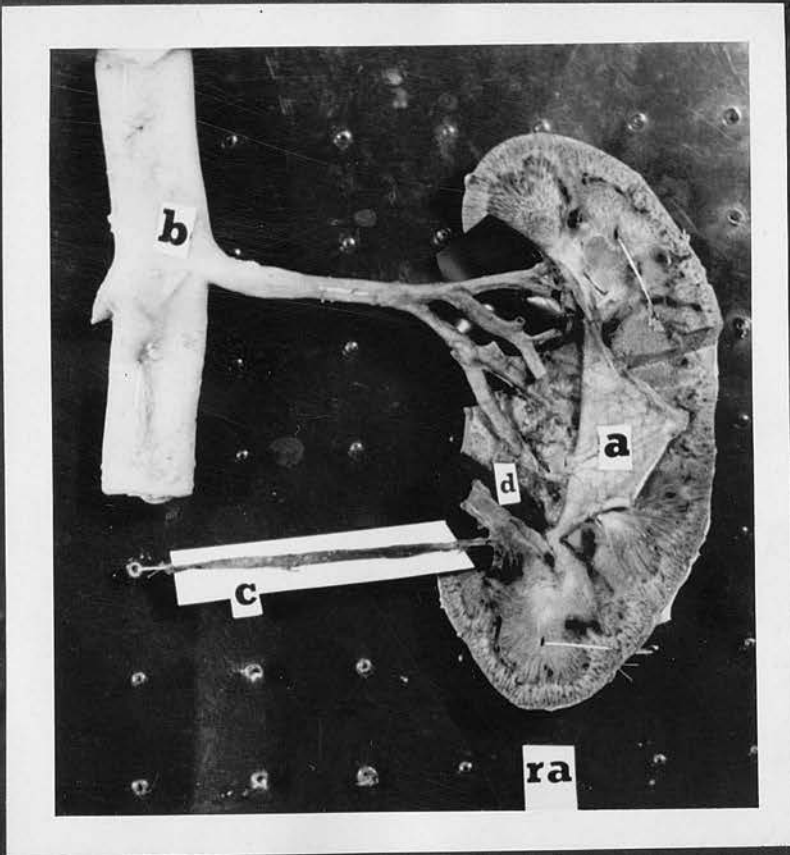
— that is 

as in the above the branching is



Parallel offshoots

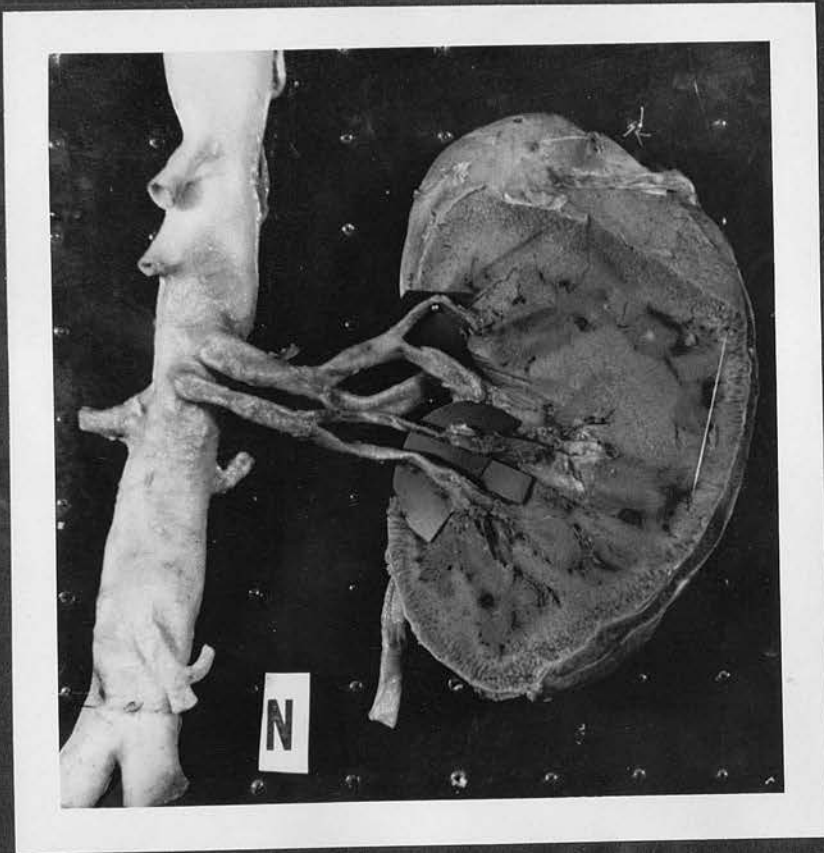
B shows aortal cephalic accessory type
also suprarenal offshoot coming in.



Type of accessory Renal Artery.

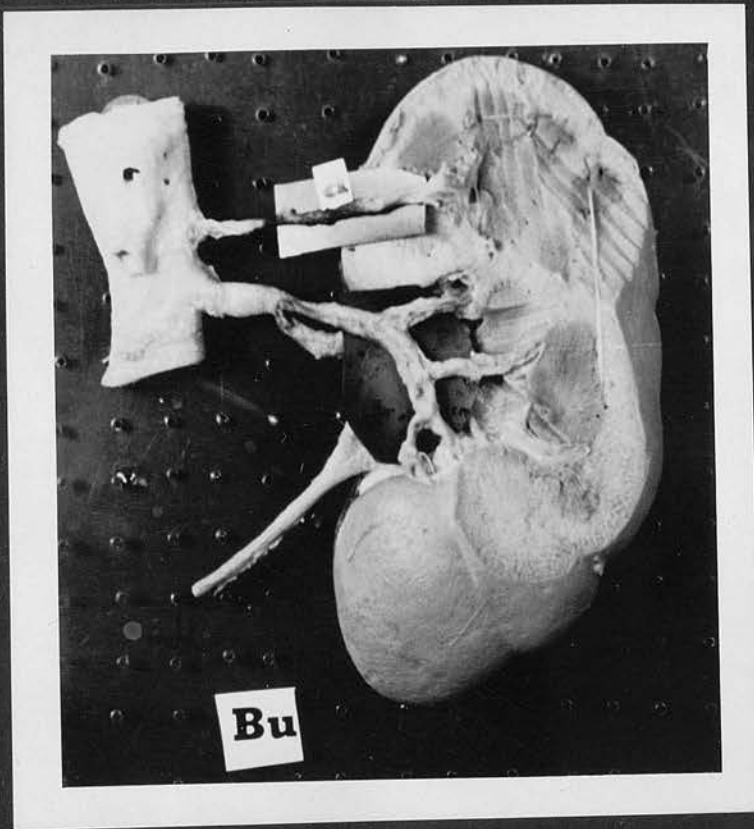
Here the main renal artery B is given off high in relation to the kidney and is partly polar.

a = is part of ureteric pelvis reflected to show a small branch c which came from Aorta to join stem d of the main artery.




Type of Accessory Renal Artery

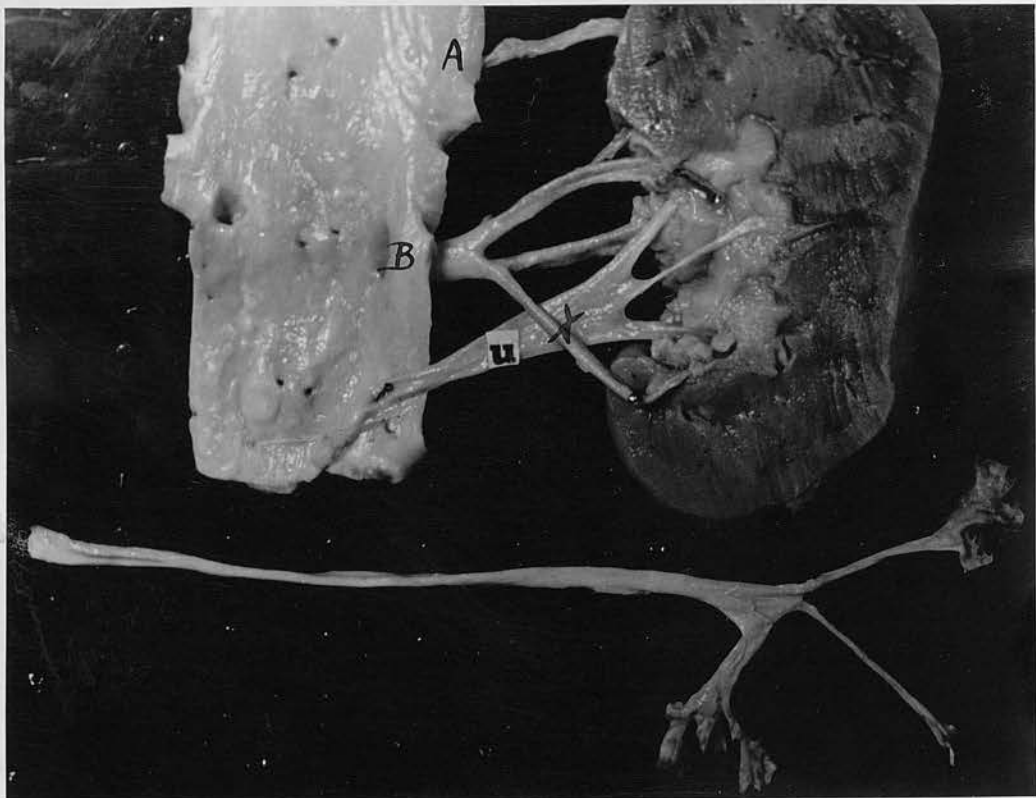
Here we have two parallel supply vessels to the Kidney — the upper being the main. The accessory is pelvic (aortic pelvic).



Type of Renal Accessory Artery

Here again are two parallel supply vessels but as compared with the previous specimen N the lower artery is the main and the upper an aortal cephalic type.

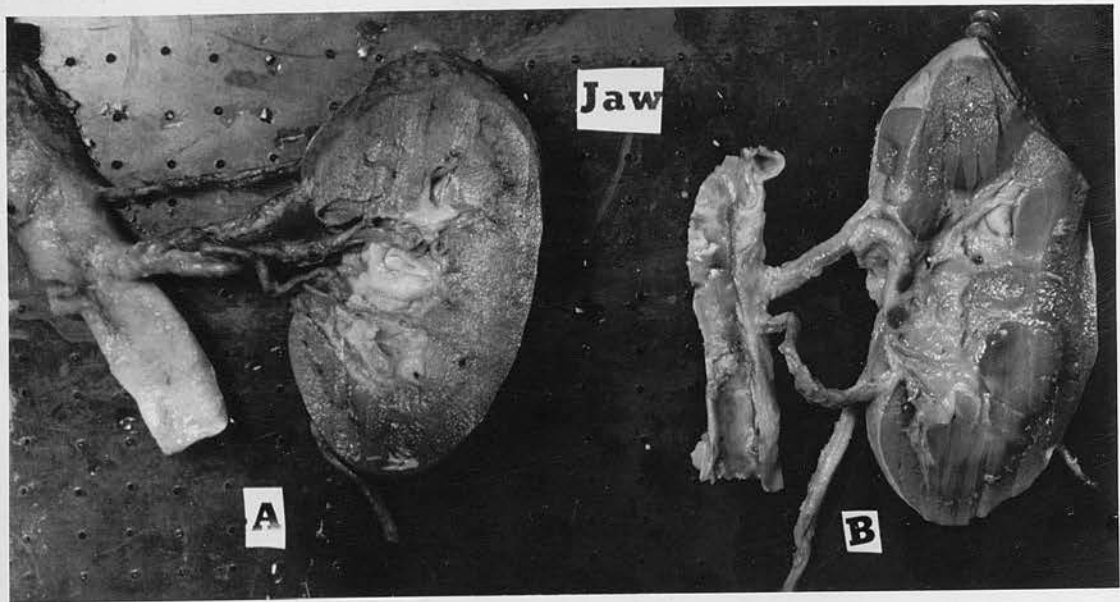
Note again the branches of the main branch — coming off the curve. in a very common way of renal branching — that is and not .



Type of Renal accessory artery.

Here the accessory springs at A to enter upper pole.
(aortal cephalic type)

B is the main artery and its lowest branch cuts across an ureter which divides outside the Pelvis. The ureter has been pinned to the Aorta to show its division. Its companion ureter is shown below. The branch X did not interfere with urine flow.



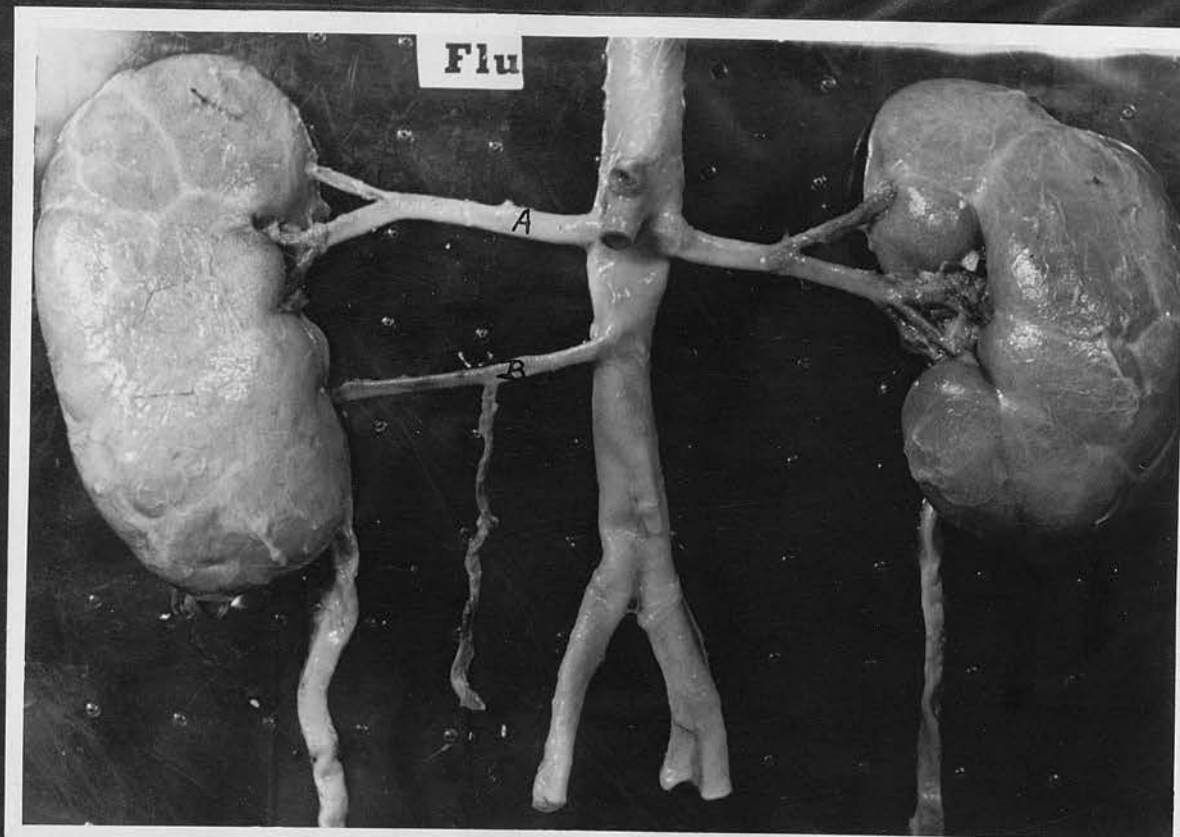
Types of Renal accessory artery.

A

upper artery is accessory and is an aorto-polar. (cephalic).

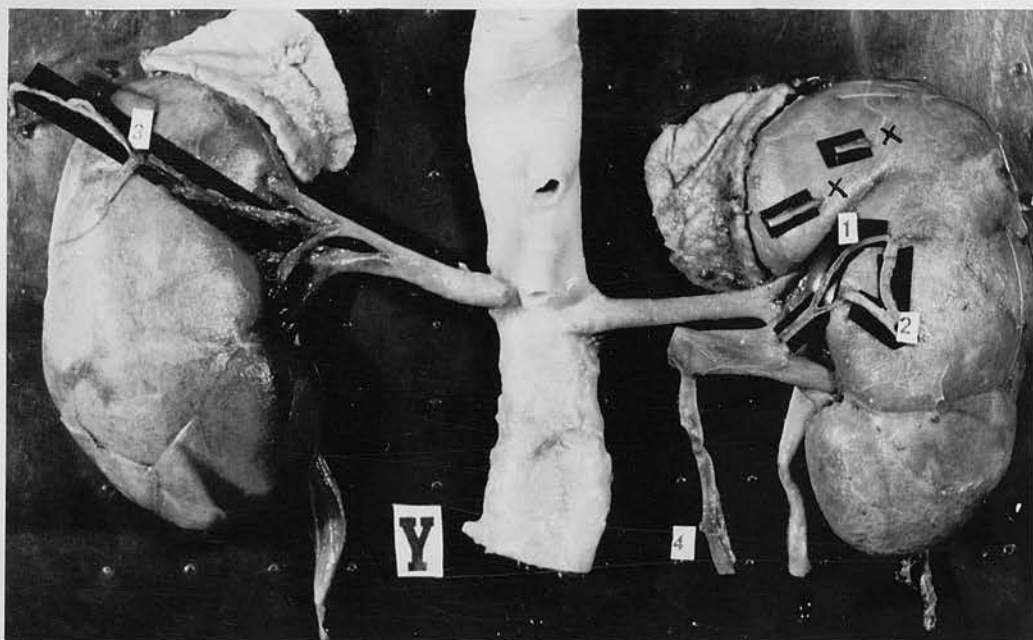
B.

Lower artery is accessory and is aortal-caudal mostly.



Type of accessory renal artery.

In this right kidney A is the main renal artery which is a very poor one and high in its distribution - in fact its distribution is what one expects from an accessory. B is the accessory with a large branch going down at right angles - its full course could not be followed due to disease disruption. The right kidney was generally poorly supplied. There was no increased pressure in this case.



Types of Renal Fascial accessory vessels.

Here on both sides we have vessels coming from the renal cortex to fat and fascia around demonstrated.

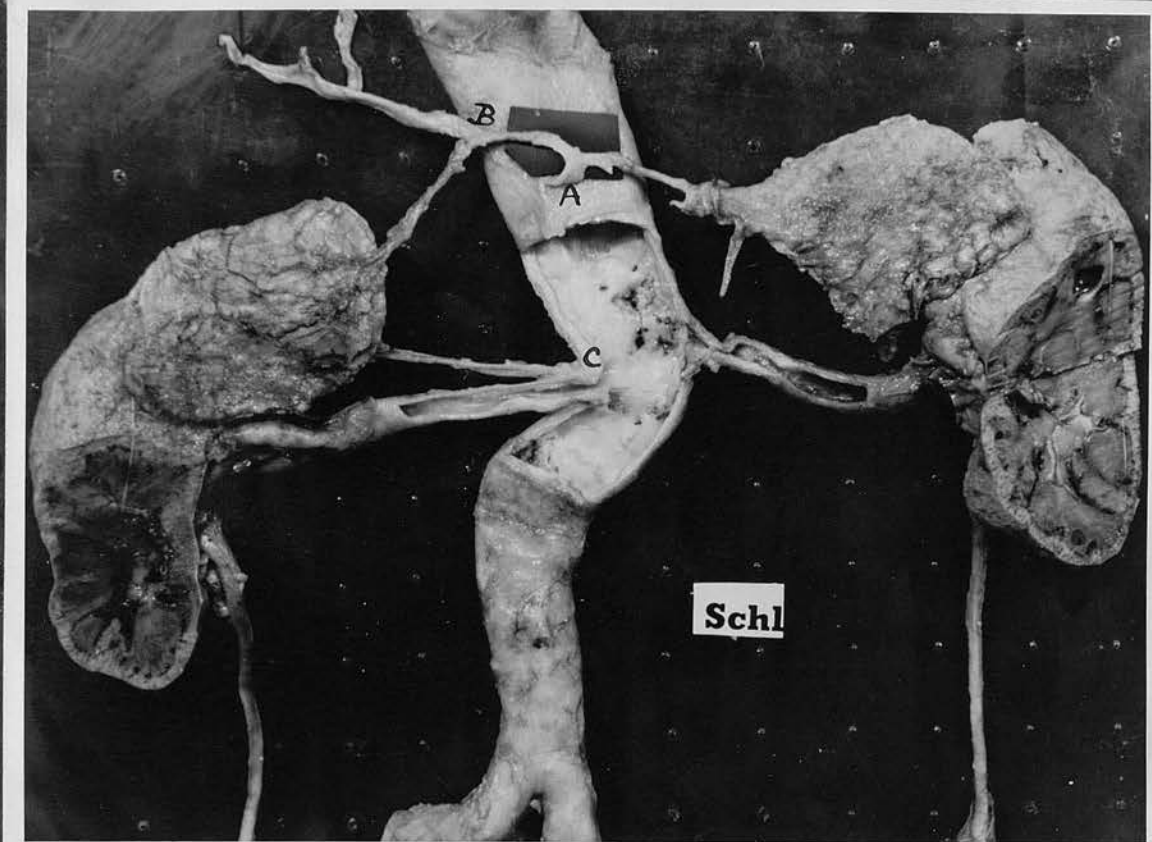
On the right (3) a branch of the renal artery comes out over cortex divides into two - one going into kidney other to fascia to form anastomoses there.

On the left (1) shows branches from Renal artery going into cortex. (Renal cortical)

While at (2) mixed in and out vessels are seen.

At X and X to other out branches are seen.

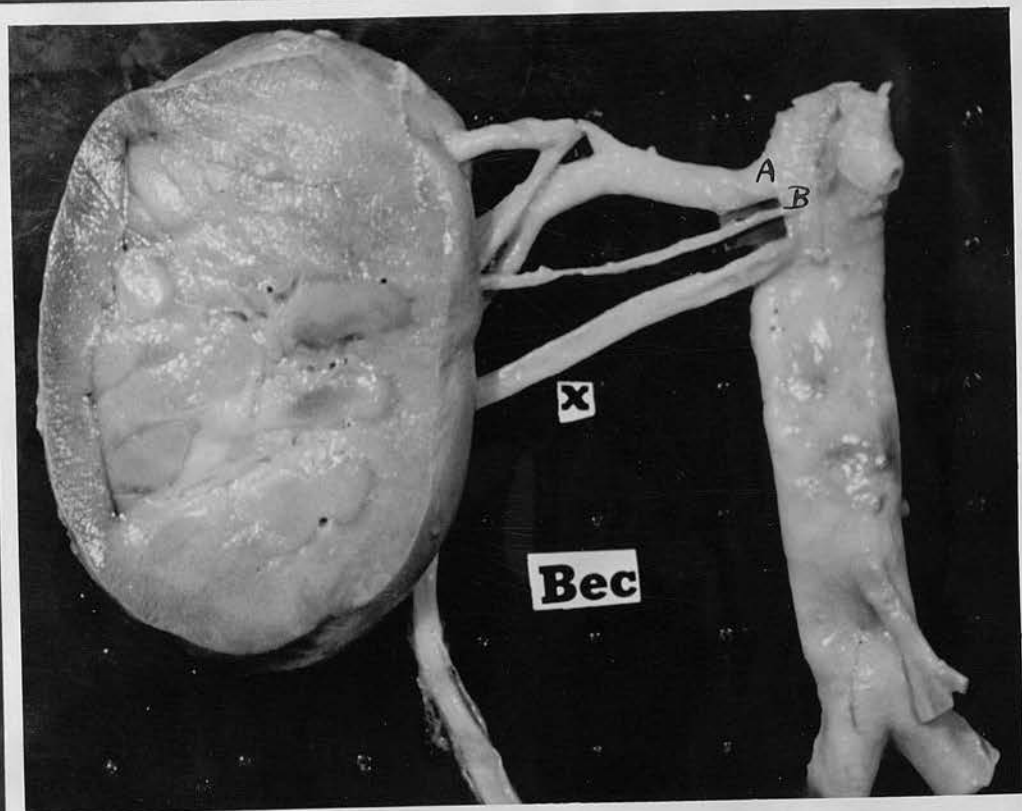
(4) is the ovarian vein.



Types of Renal accessory artery.

On the right an aortal polar accessory is seen at C. Its mouth was obstructed due to bad aortic disease. There was no high blood pressure in this case.

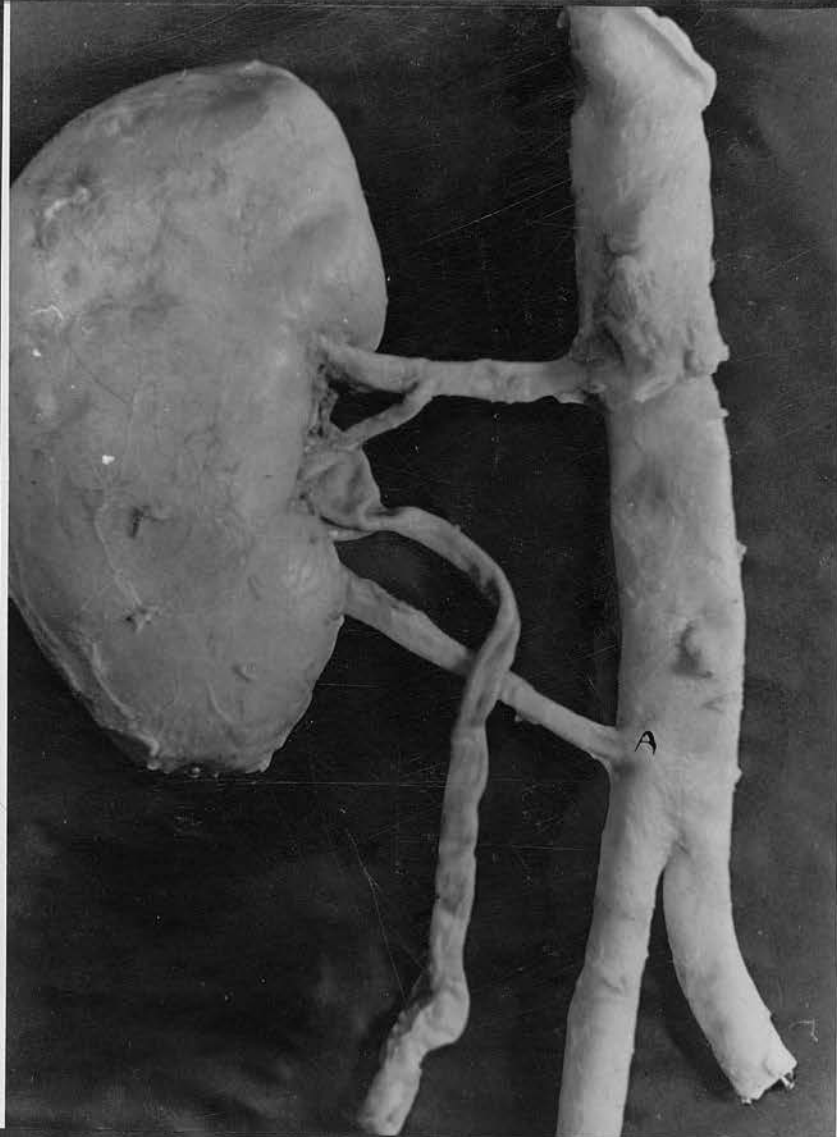
At A we see a common stem divide into two suprarenal vessels — a branch of one of which divides into a small suprarenal and a large vessel of supply to the subphrenic area.



Type of accessory renal arteries.

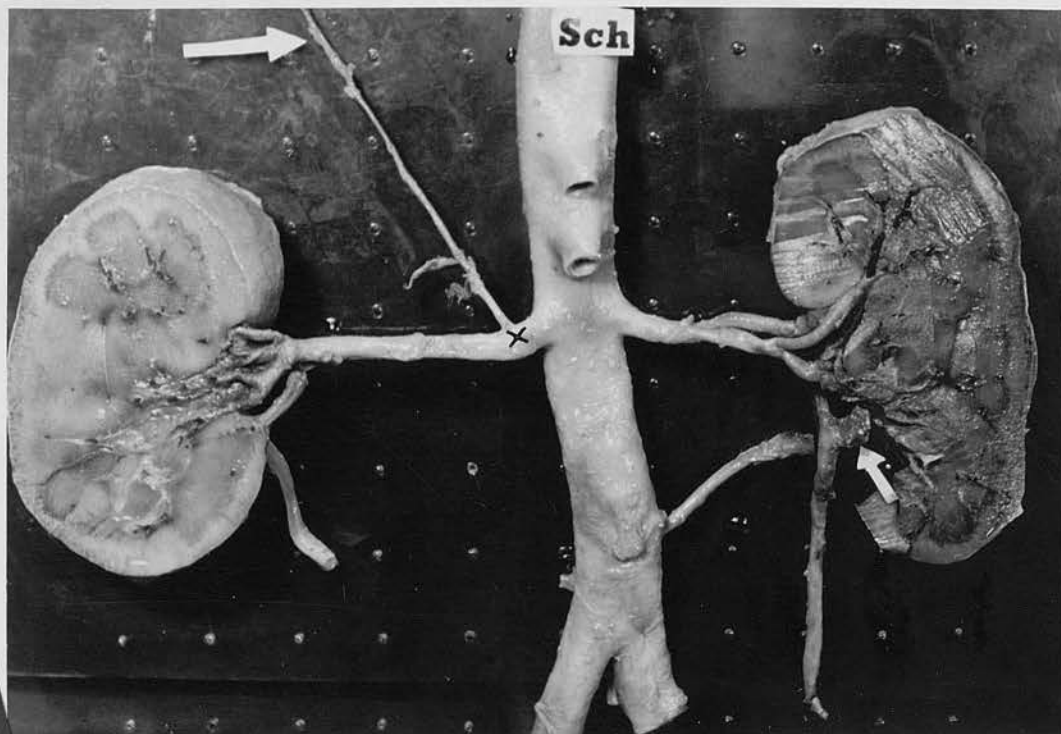
Here the main artery is high and branches more into the upper pole than the pelvis.

Immediately below a small accessory B arises to go into Pelvis, while lower at X a large smooth branchless vein like accessory goes from Aorta into lower pelvi-polar area.



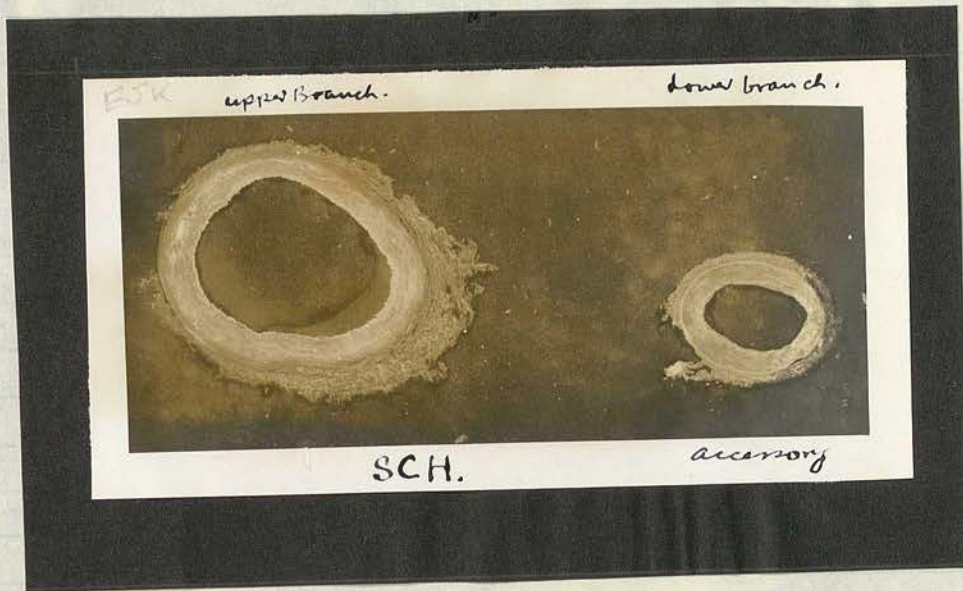
Type of accessory Renal artery.

Here the ureter is in front of the accessory artery A. Usually it lies behind. This artery did not cause any trouble with the ureter. There are remarkably few large branches in the arterial supply. Here the accessory artery is a branchless vein like structure like that of the immediately preceding photograph. (Bec).



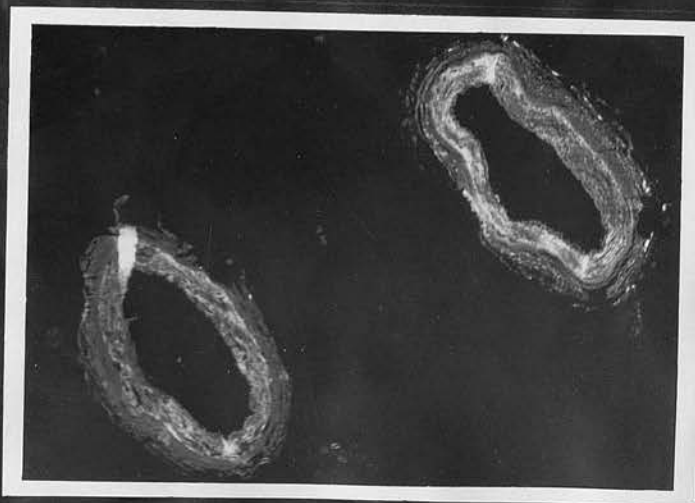
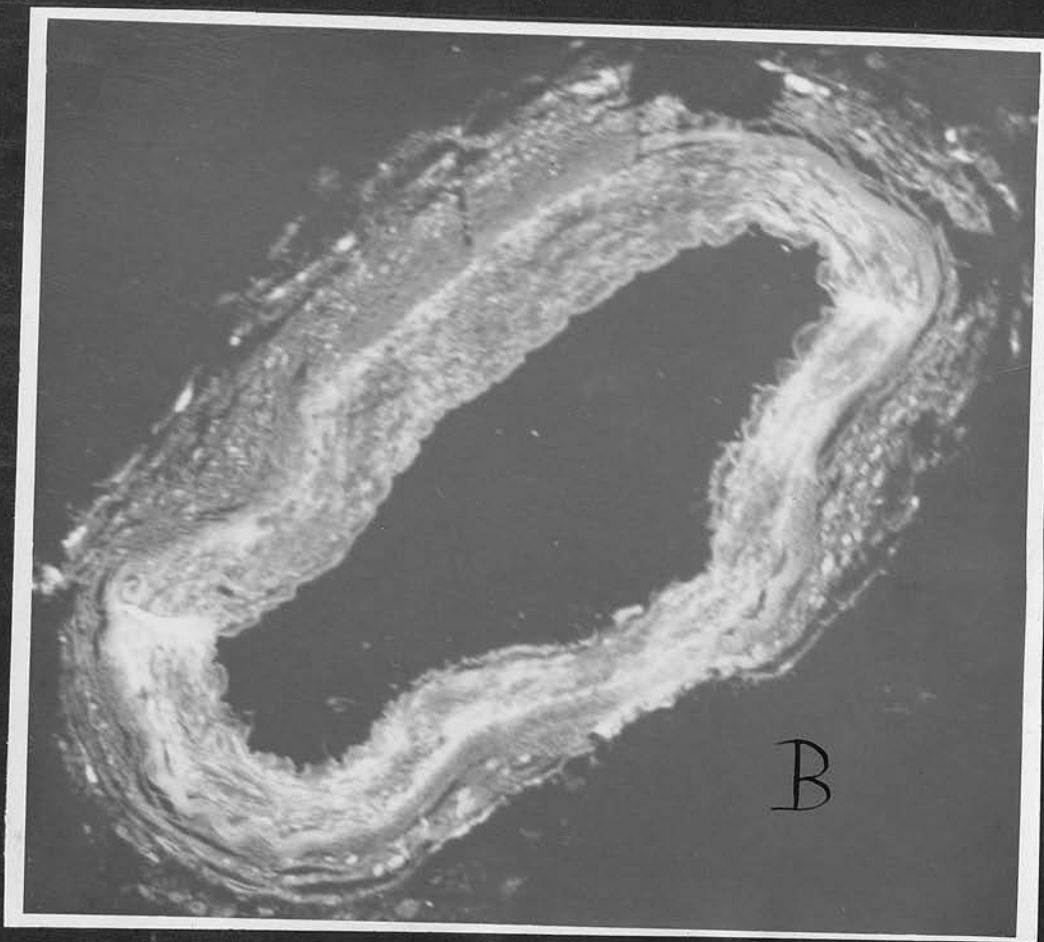
Type of Renal accessory artery

This a low type of accessory artery like the previous photograph only on the left. Also the ureter lies in front but is not affected by it. On the right the artery arising from the Renal at point X supplied ganglia, then the suprarenal then went on to a very long length to the diaphragmatic muscle area. Overleaf the two arteries on the left have been sectioned to show the coat thickness.



This photograph of a section shows the two branches going to the left kidney in the previous page. There are no essential differences except in size. Both have good muscle coats and the accessory had an important part in the supply ~~area~~ of the lower pole of the kidney.

For photographic technique see Photographic Notes at end on Page 51.



↑ A.

↑ B.

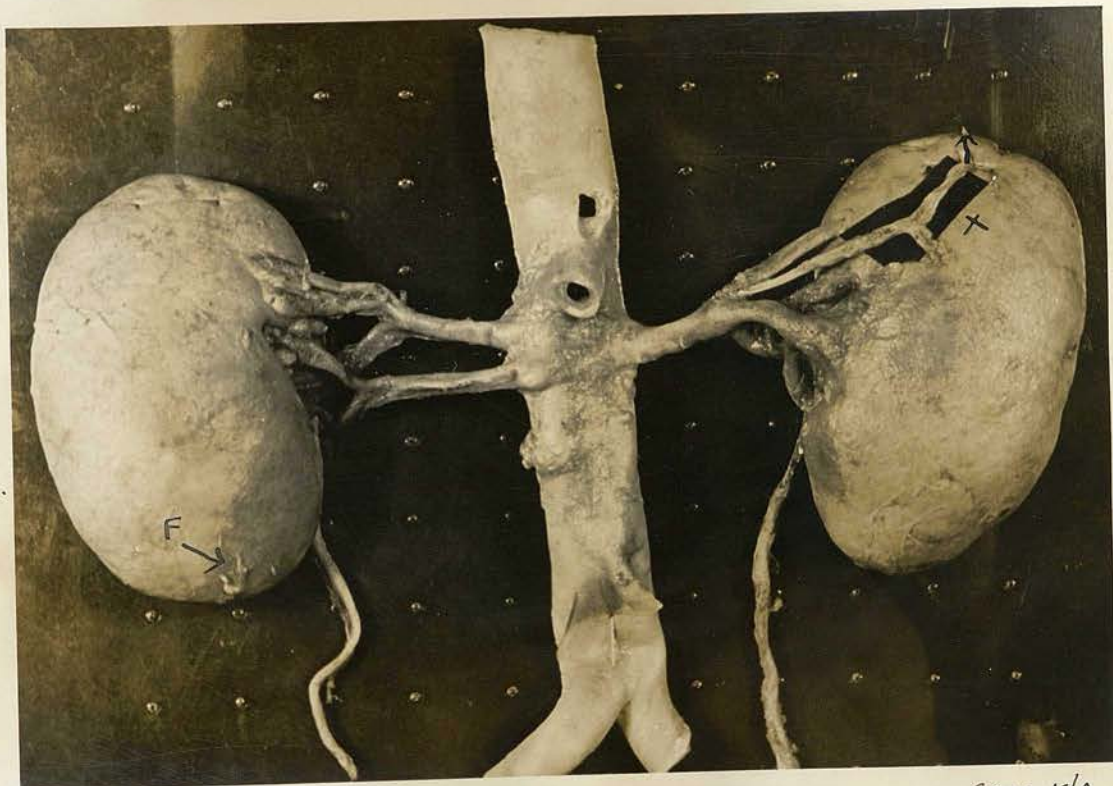
Here are sections of a typical pair of Renal arteries.

A the accessory. B the main artery.

B is enlarged in the photograph above.

Both have good muscle coats and there was on close examination no difference in constitution.

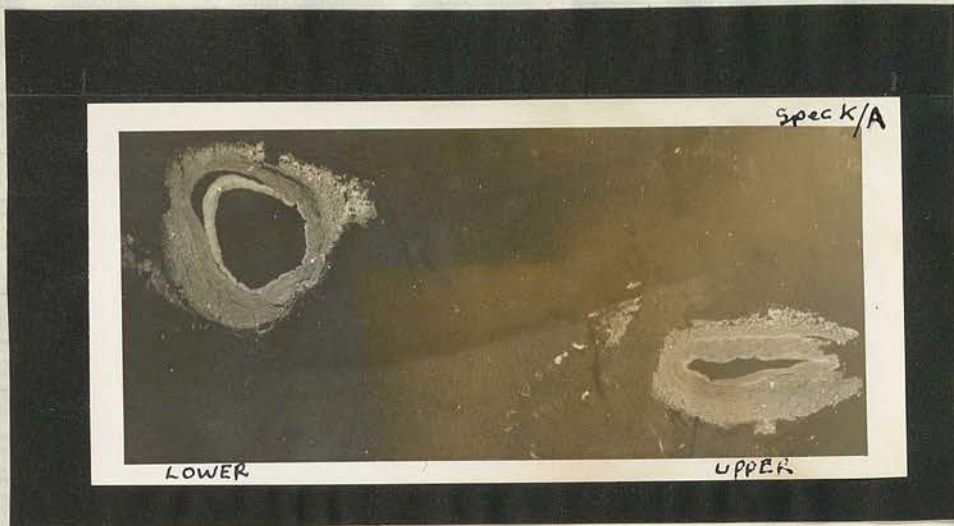
Photographic technique on Page 51.



Spec K/A

Type of accessory arteries.

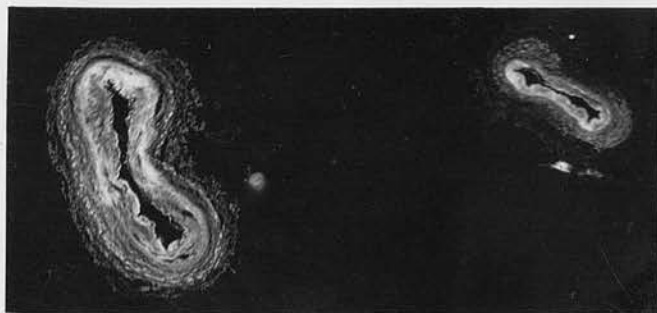
Here are two arteries on the right both springing off close to each other and making it difficult to tell which is the main artery. On section the upper artery seemed to have the firmer construction. (See overleaf)
 On the left at X are seen the division of a small branch of the renal — one branch dipping into the cortex the other going up into the suprarenal fascia.
 Another cortical fascial off shoot is seen at F →.



This is a photograph of sections of the two arteries on the right in the previous page (Specimen K/A.).

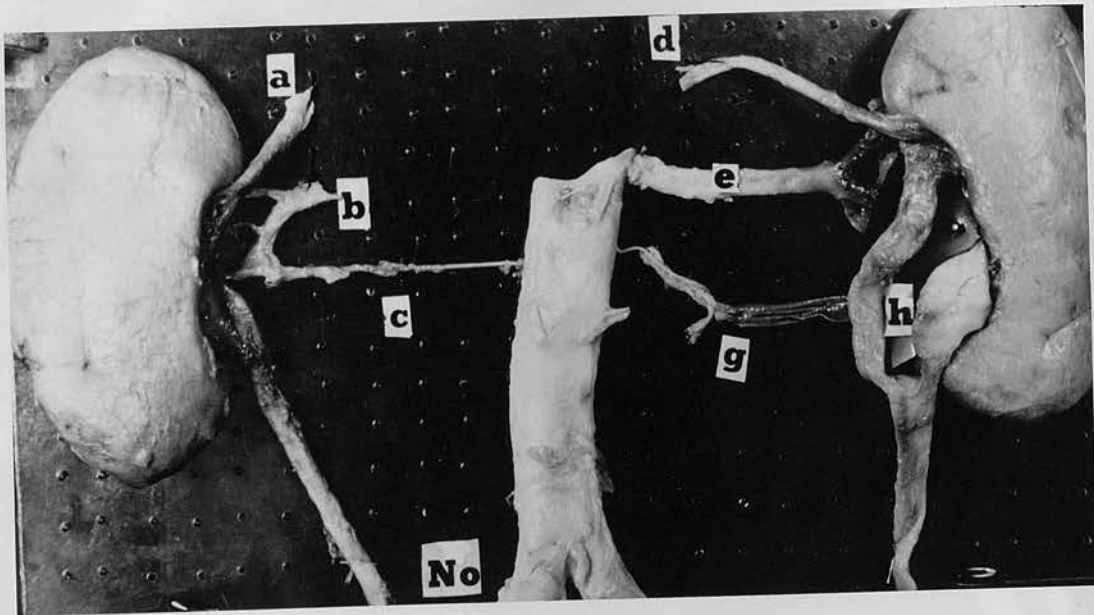
The lower artery which was probably the accessory was thinner in coat and less muscular compared with the upper which was stout and well coated though of smaller calibre.

Photographic Technique see Page 51.



"Vein" like type of branchless
accessory as in Pages
THIN WEAK COAT.

ACC

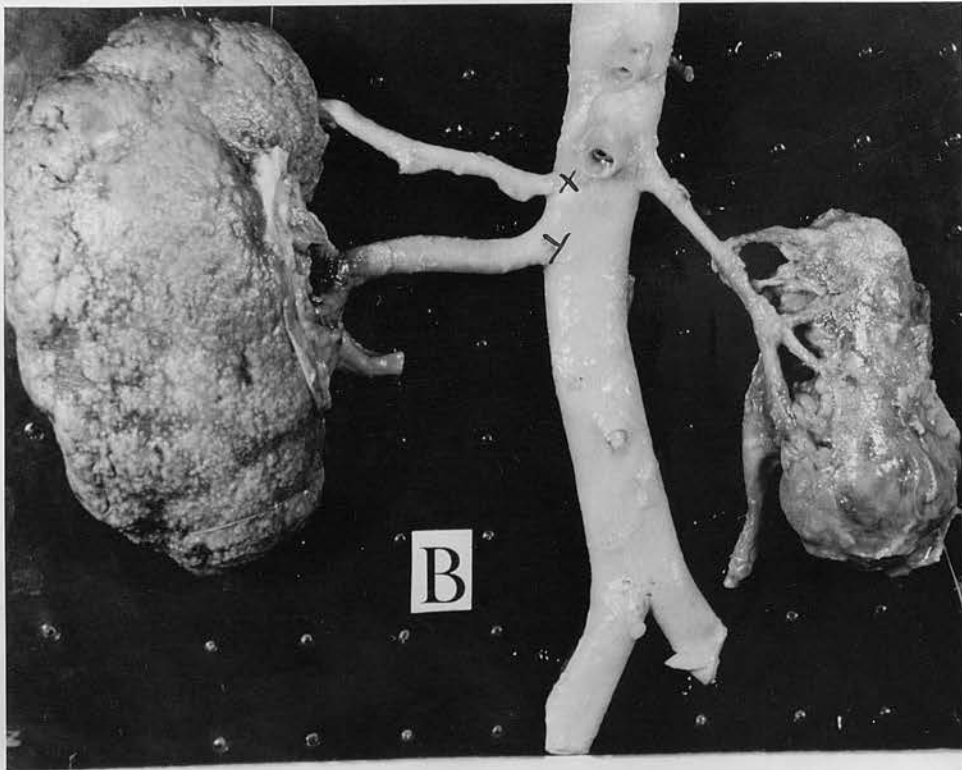


Type of Accessory Renal Artery.

Here one finds on the right an accessory Renal going into the upper pelvis (a). The main renal artery b. At c we find a very thin renal artery corresponding to g on the left. On the left e is the main artery while d is an accessory corresponding to a. Notice on the left h the large abnormal bifid pelvis of the ureter. The part played by the accessory arteries in interference with the ureter is discussed in the Thesis. The arteries on the right were cut from origins to study their coats.



The above ^{shows} ~~the~~ ^a ~~the~~ sections of the two arteries — main e and accessory d from the left side of the previous page specimen (NO). The main artery e had part notched away for identification purposes. Here the accessory d seemed to be sturdier and better muscle coated than the main artery.



Type of accessory Artery.

Here is an aortic cephalic type of accessory renal on the right X.

It is associated with a very late branching main artery.

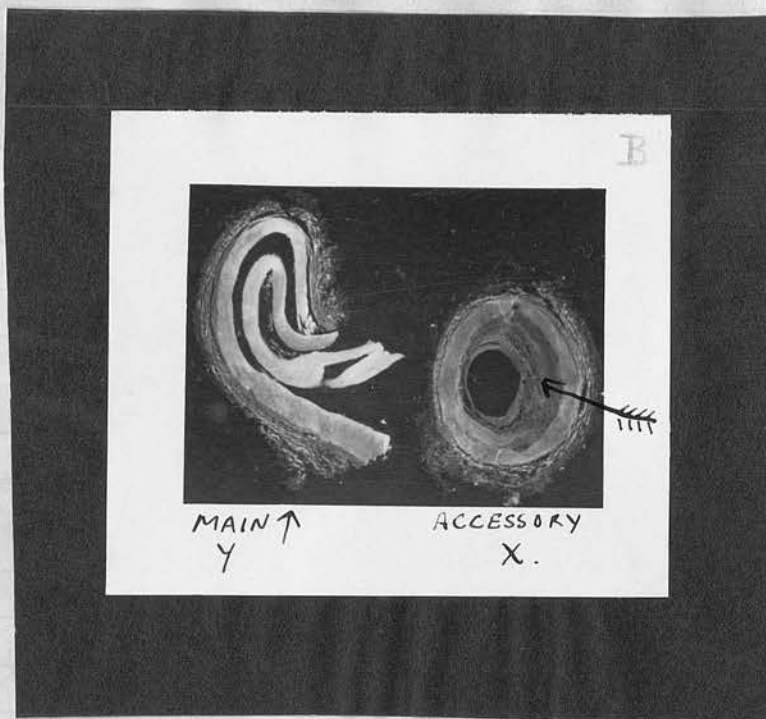
On the left the Kidney was low and rudimentary.

Here there was raised blood pressure and renal failure.

The granulation of the Kidney surface is well seen.

A section of the main artery Y showed changes.

See over leaf.



Sections from two arteries in Kidney shown in
previous page.

The person died from high blood pressure and renal failure — the left Kidney being practically functionless and small.

The arteries on the right side were both diseased — the main artery Y being remarkably so.

The coats seemed hyalinised.

The accessory artery X had also much coat disease.

Springing from "See arrow" on right.

Two only accessories are seen on right but they spring

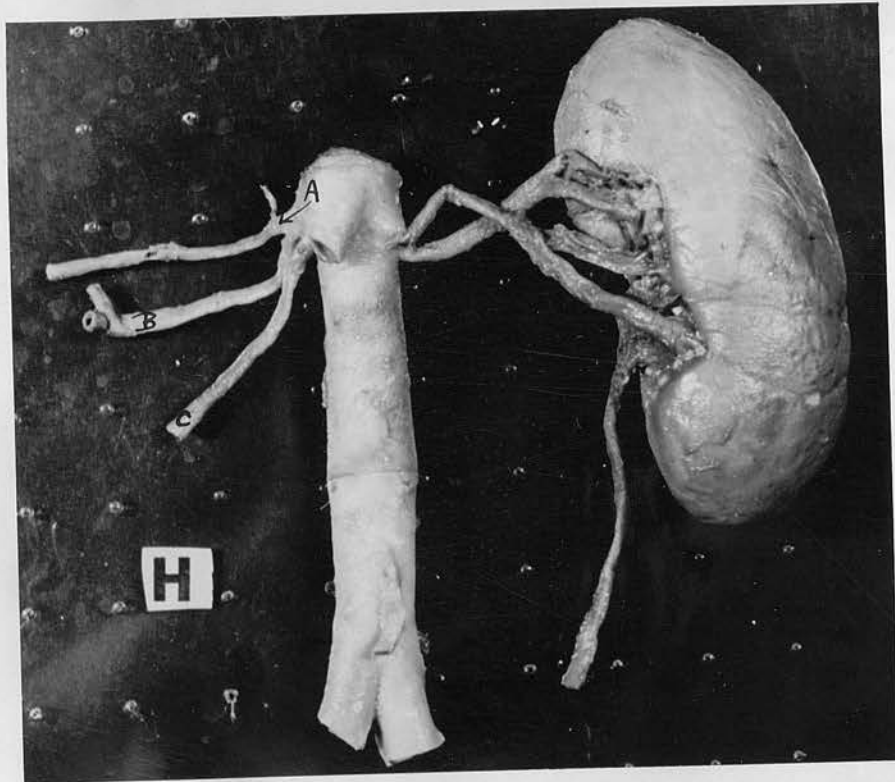
from the same place. A on the right was capitate

B was pituitary and a third pituitary

The two arteries on the left were normal

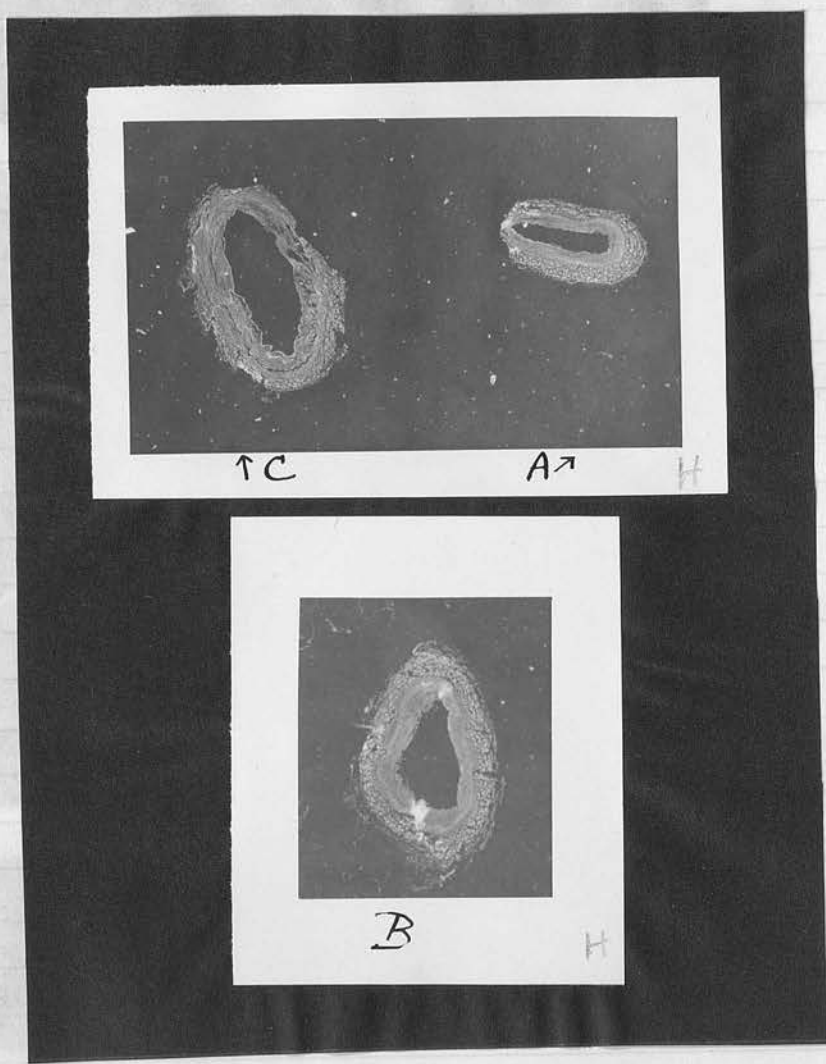
after the end

See Page 51, for photographic Technique. 51

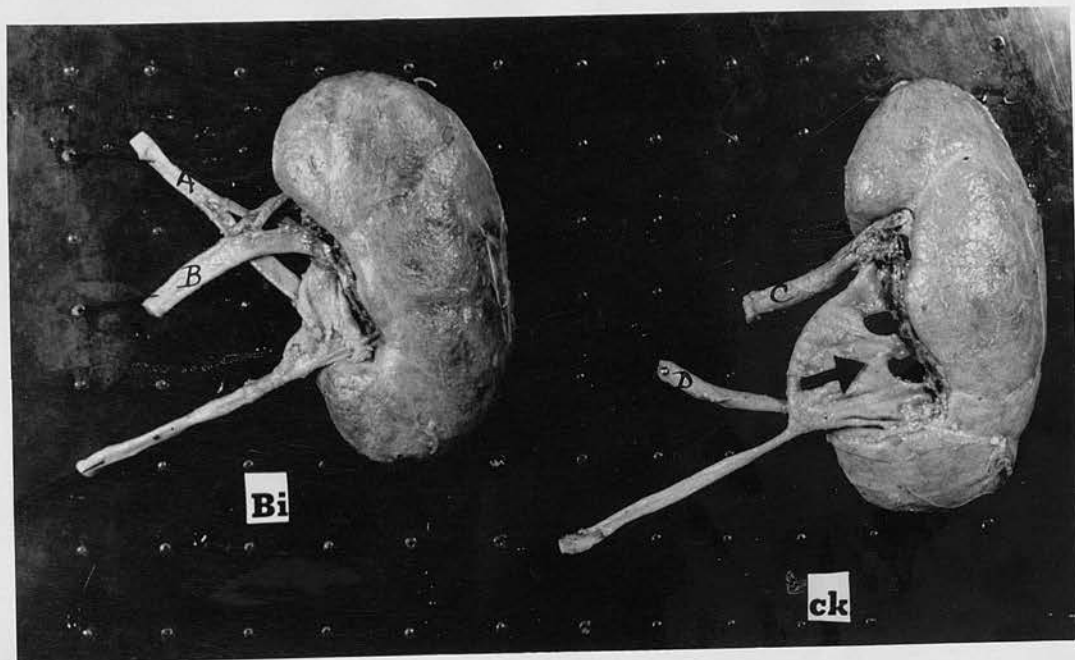


Types of accessory arteries.

On the right A, B, and C we have three accessory arteries springing from the same place, while on the left two only accessories are seen — but they spring from the same place. A on the right was cephalic, B was pelvic and C lower pole caudal. The two arteries on the left were crossed as is often the case.



Sections of the three arteries in specimen H previous page. These three arteries - B being the main one - showed no difference in structure - merely difference in size. They all contributed to the blood supply almost equally. The upper A left took part in the supply of the suprarenal gland.

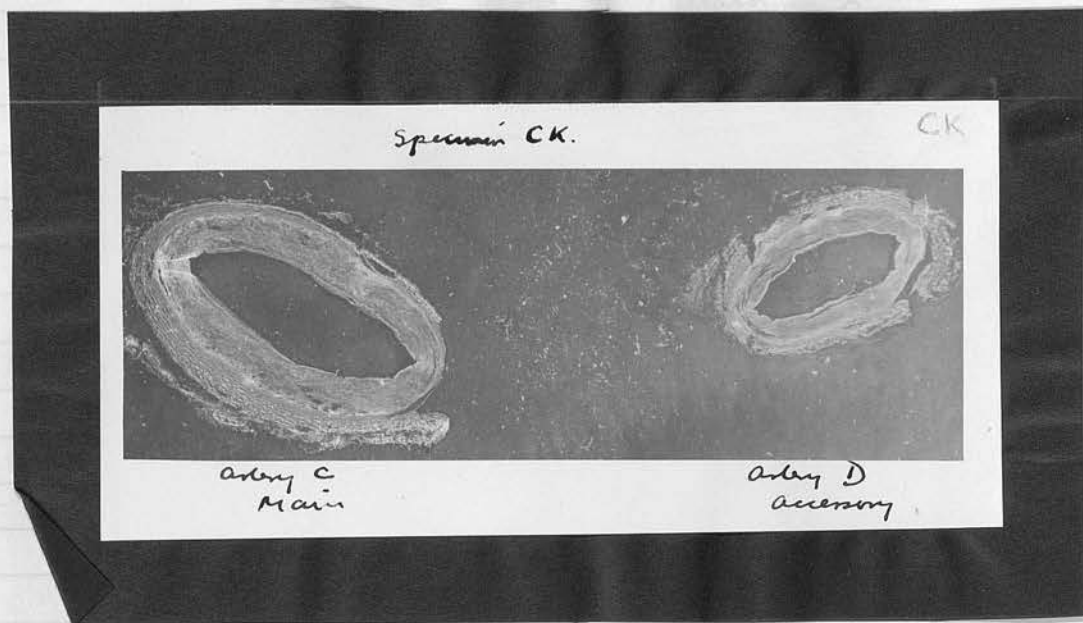


Types of enlarged ureteric pelvises.

Here in specimen Bi we have an enlarged pelvis of the ureter with no accessory artery within reach. A and B show the accessory and main arteries respectively.

In specimen CK we have again an enlarged pelvis of the ureter and behind an accessory artery D. On carefully study of the relations in situ there was no evidence that the accessory artery had anything to do with the enlargement.

C is the main artery.



Here are sections of the main (c) and accessory (d) arteries of the kidney CK. Both had good coats — both had some disease — but careful study showed no special difference of the main from the accessory. Both had important parts to play in the blood supply of the kidney.

PHOTOMICROGRAPHIC NOTES.

① The two silver stained arteries on Pages 5 and 6.

Camera = Leica with Micro-Iso-attachment.

Film = Kodak Panatomic X.

Filter = green.

Exposure = 1 sec.

Magnification = $\times 425$.

Developer = Kodatol Fine Grain D K20.

② The sections of vessels on Pages 38. 39. 41. 42
44. 46. 48. 50.

(a) Stained with

Phosphomolybdic acid 1 gramme

Water Soluble Blue 0.5 "

Orange G. 2 grammes

Water 100 cc.

Stained Slides put in Leitz Valoy enlarger and
magnified image thrown direct on to
Kodak Nikko Extra Contrast paper — thus
giving an enlarged negative print with
enough differentiation to show coats.

Developed in Kodak X-ray D 196.

Magnification mostly $\times 7$.



Statistics

Out of 400 pairs of Kidneys over 90% with
accessory supply.

Distribution of accessory supply.

Accessory Supply on BOTH SIDES	25%
Accessory Supply on RIGHT SIDE ONLY	15%
Accessory Supply on LEFT SIDE ONLY	50%

The Anatomy of the Ovarian Veins.

Two cases illustrating
variations
in

The termination of the Ovarian Veins.

One NOT published before.

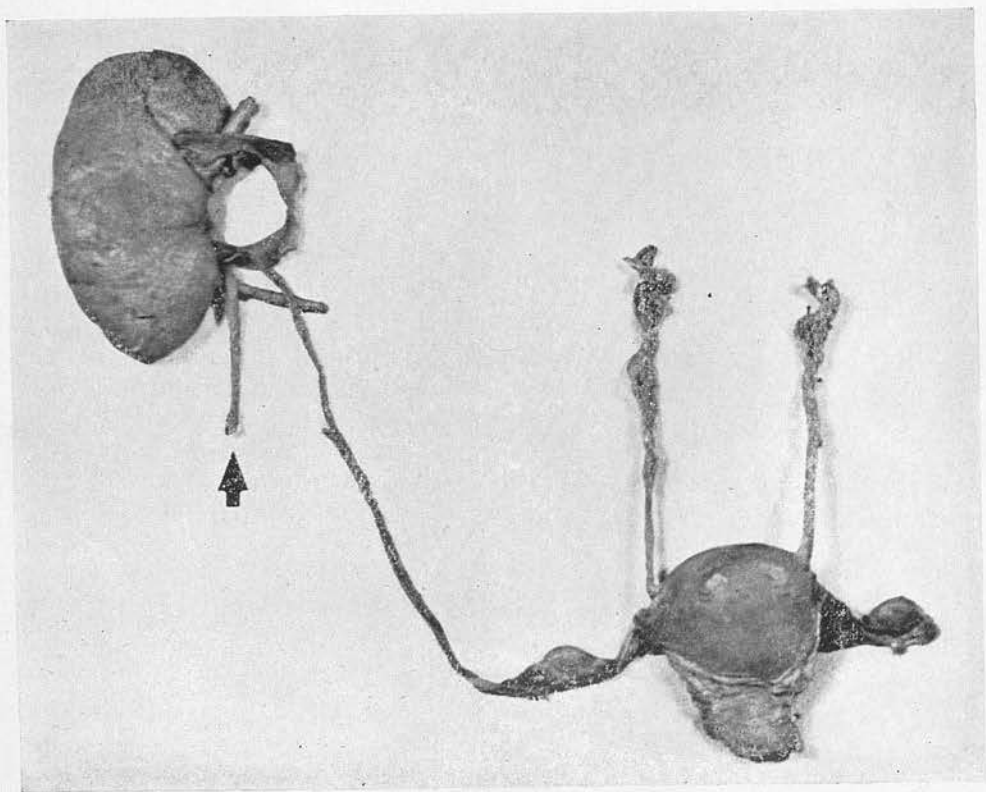
One published first by myself.

Reprinted from

The Journal of
Obstetrics and Gynaecology
of the British Empire

Volume LI, No. 6, December 1944

SHERRATT AND HUGHES
MANCHESTER



A.R.

Transposition of the Ovarian Veins

BY

ARWYN ROBERTS, M.B., Ch.B.

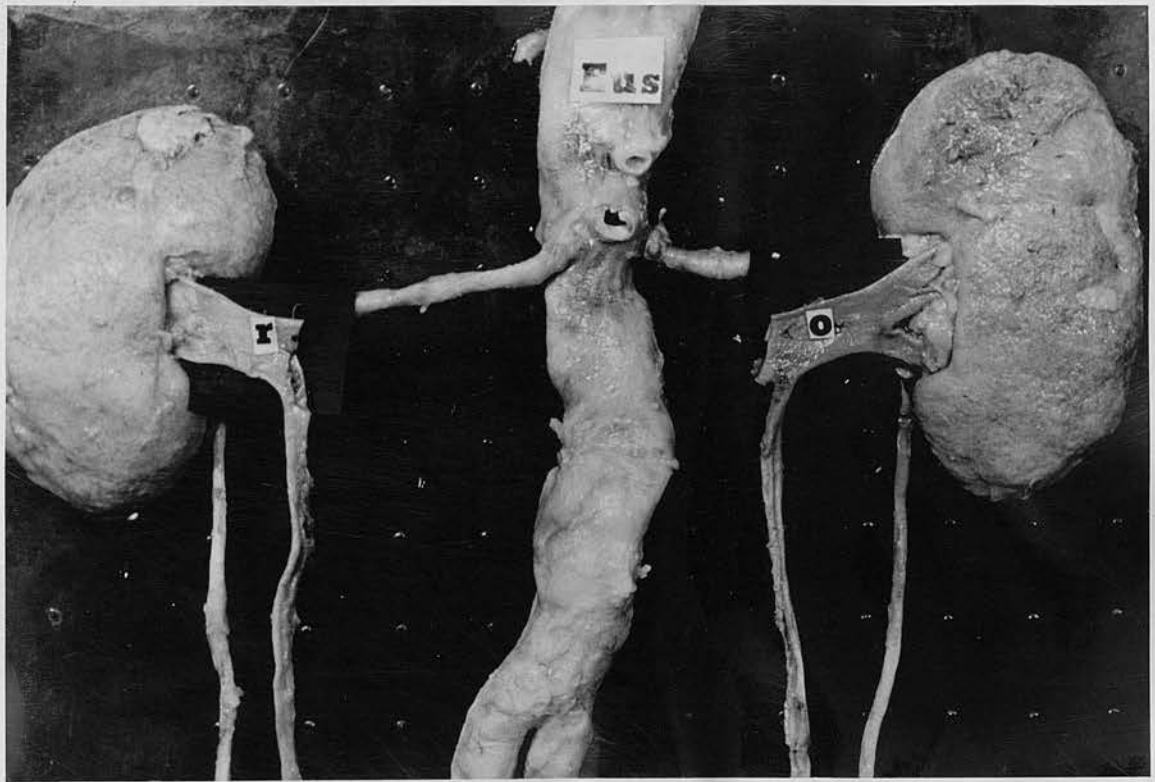
Pathological Department, Runwell Hospital, Nr. Wickford, Essex.

IN text books of Anatomy and Gynaecology the right ovarian vein is stated to enter the vena cava, and the left ovarian vein to enter the left renal vein.

These veins are long and frail, and in postmortem and anatomical dissections are apt to be torn or dragged from their origins or terminations, so that a proper study of their relations cannot be made.

I have found as the result of many careful dissections that in a very small percent-

age of cases there is a transposition of the ovarian venous termination, as seen in the photograph where the right ovarian vein enters the right renal vein. There is in this case an abnormal renal artery also which enters the lower pole of the kidney behind the transposed vein and the ureter. The ureter is marked with an arrow. For permission to publish this note I am indebted to Dr. R. Strom-Olsen, the Physician Superintendent, and for the photograph to Mr. T. C. Hall, M.S.R.



In the preceding page transposition of the ovarian vein is shown. This happens in a very small percentage of cases.

Here is shown a case where both the ovarian veins go into the renal veins.

This happens in a still smaller percentage of cases.

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Oliver, J. and Lund, E.M.: Plastic Studies in Abnormal Renal Architecture, Arch. Path., 15:755, 1933.

Shaw, H.T.: Hyperpiesia and Hyperpiesis, London, 1922.

Part 2 of thesis.

Observations on the anatomy of the Coronary arteries with Reference to their Pathology.

Observations on the anatomy of the Coronary Arteries.
with reference to their pathology.

Another perplexing aspect of coronary thrombosis is the frequency with which a particular part of the Coronary system is involved. A most common site is in the descending part of the left coronary artery about one inch from it's origin - that is the first inch of the anterior descending branch. Sclerotic changes and thrombosis are so common at this particular spot that it makes one suspect that a mechanical factor is involved.

(Levine.S.A. Clin Heart Dis. Saunders. 1936.)

The descending anterior branch of the left coronary is the one which is most commonley the seat of disease.

(Shennan.T. Post Mortems and Morb Anat. Lond. 1935.)

The most common site of coronary thrombosis is in the first three centimeters of the ant descending branch of the left coronary artery.

(Ross.J.M. Post Mortem Appearances. 4th Edit. 1941.)

-Atheromatous narrowing of coronary commonest in the ant descending branch just beyond it's origin.

(Muir.R. Pathology. 4th Edit. P 345. 1936.)

Thrombosis- most often found in the main descending branch of left anterior vessel.

(Dible and Davie. Pathology. P 55I. 1939.)

Vessel most commonly affected is the descending part of the left coronary- the first half inch.

(Beattie and Dickson. Pa thology. 4th Edit.P 467.1943.)

The above quoted authors and many others testify to the prevalence of left coronary artery disease in the first part of it's anterior descending branch. Many studies have been made of this particular branch- injections, c ross sections, cholesterol studies, et cetera.

Levine in the first of the above quotations hints at a mechanical cause, and from studies of several hundred dissections of the vessels I am of opinion that the pattern of these arteries can be resolved into two main types- mechanically sound and mechanically unsound. I a m also of opinion that such patterns whether good or bad are capable of being inherited, in the same way as many other anatomical traits are inherited.

The left coronary artery is a large vessel, and it's ramifications are a brubt as compared with the right, and such ramifications have amongst them deep penetrating branches fundamentally designed to nourish a larger muscle mass. The artery lies partly free and partly on the

surface, and is fixed at the site of each penetrating branch and buckling tends to take place with regions of increased stress at bends thus favouring the development of atherosclerosis and thrombosis.

Once damage has been done in a mechanically strained part, then cellular infiltration, vascularisation, proliferative changes, deposition of lipoids, calcium and soaps take place. The thickening of the intima causes lumen narrowing, while a thinned media with haemorrhages into it cause more weakness with eventual aneurismal change or rupture. —→ Photo Page 3.

Obstruction in many cases seem to be caused by small haemorrhages which seem to be the sequelae of capillary vascularisation of the wall. Also these intimal haemorrhages can push atheromatous plaques into the vessel lumen or destroy endothelium to cause obstruction and thrombosis.

The pathological changes above described have been well described by many, some of which are quoted below, so I propose to go on with the mechanical causes of these changes.

References for pathological changes in walls.

- Kirch.E. 1930. Ergebn allg Path Anat 23 427.
Wartman.W.B. 1940. Am.J.Med.Sci. 186, 27.
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1940. Am J Med Sci. 13. 122.
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Wartman W.B. et Alia. 1938. Biology of Arteriosclerosis.
Nelson M.G. 1941. J.Path Bact. 53, 105.

An example of a much diseased portion of the first part of the descending ant branch of the left coronary is seen sectioned and stained with Haematoxylin and Eosin in Plate One Page Three .

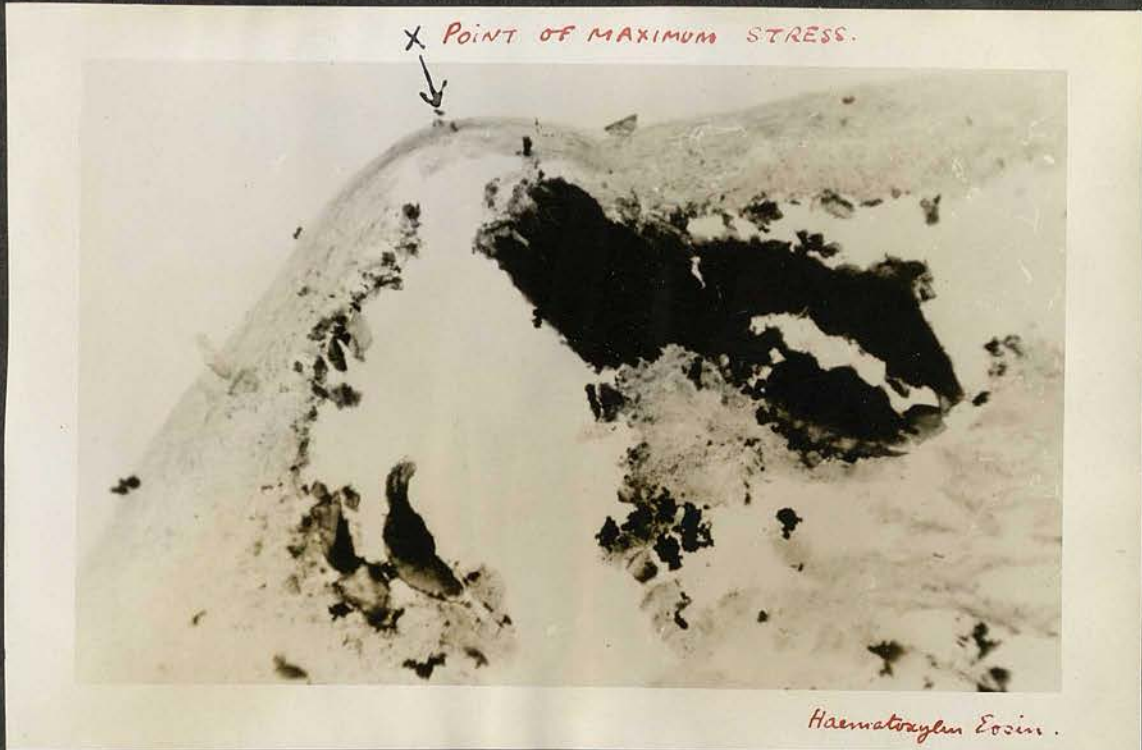
Before we study the mechanical aspects of the left Coronary artery let us first glance at it's usual normal anatomy.

The usual course of this artery is from left sinus aortae forwards between Pulmonary artery and left auricle to upper end of anterior longitudinal sinus, where it divides into two main branches,

(1) Ant descending branch which proceeds apexwards in anterior longitudinal sinus to anastomose with an inferior branch of right coronary.

(2) Circumflex branch which runs in coronary sinus to anastomose with circumflex branch of right coronary.

Both give early small branches to valves and great vessels which act as tugging mooring ropes to cause stress on the parent vessels.

PLATE ONE

Section from an anterior descending branch of left coronary artery at point where it bends to be more firmly fixed on and in heart muscle. That is point C in previous diagrams. The angulation effect is seen and the coat is diseased nearly right through and much black deposit of calcium (dark blue in stain).

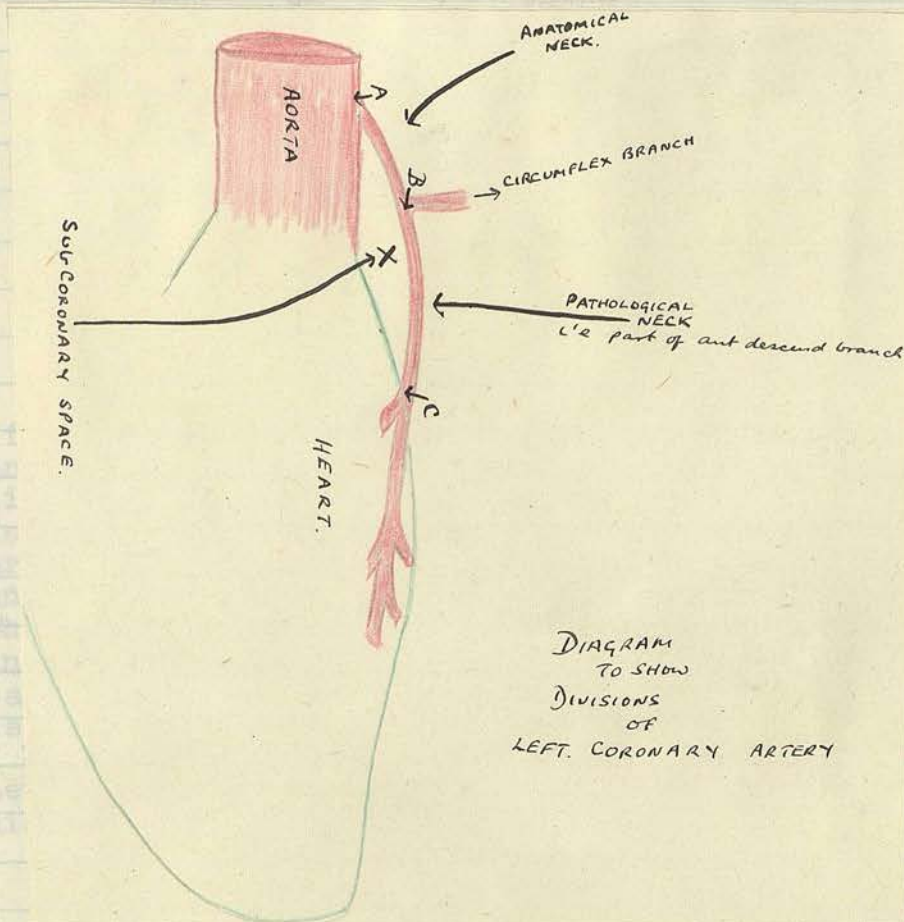
(See photographic notes
at end)
(Page 12)

To help in the study of the pathology of the left coronary artery and it's descending branch I have used the following three new terms. Just as the Humerus has an anatomical and a surgical neck, I describe the left coronary artery as having an anatomical neck and a pathological neck.

The anatomical neck I define as the part of the coronary artery from its origin in the aorta to the part where it divides into circumflex and descending branches. AB in the diagram below.

The pathological neck I define as that part from the origin of the anterior descending branch to the part where the ant desc branch becomes firmly fixed to the heart muscle either by binding or by the sending down of a deep muscle branch, or as is usual by a combination of both. This is BC in the diagram.

Another important term I have coined is the "sub coronary space". This is the space marked X in the diagram, and is defined as the space between the coronary vessels on one hand and the aorta and the heart on the other hand. In this space there is only loose fat and fibrous tissue and the coronary vessels are unsupported here. Pieces of paper are seen thrust into this space in the heart photographs.



The anatomical neck may be long or short, the pathological neck may be present or absent. If the anatomical neck is long the pathological neck is absent and this is the type least prone to serious pathological diseases.

In other words the first inch or so of the anterior descending branch may take part in forming the unsupported arch over the subcoronary space or not. When it does form part of the arch it is subjected to stresses and strains which in the greater percentage starts serious pathological changes.

These stresses are mostly about point C in the diagram → *Diagram 2* below, i.e. in about the first inch of its course.

The stresses at C are at the fixation point of the artery and are mainly due to the fact that one part, (the proximal) has greater play from aortic and ventricular pull and is part of an unsupported pulsating arch; while the other part (the distal) is fixed to the heart surface and has much less play. At this point C there is also angulation to help in accentuating the stress.

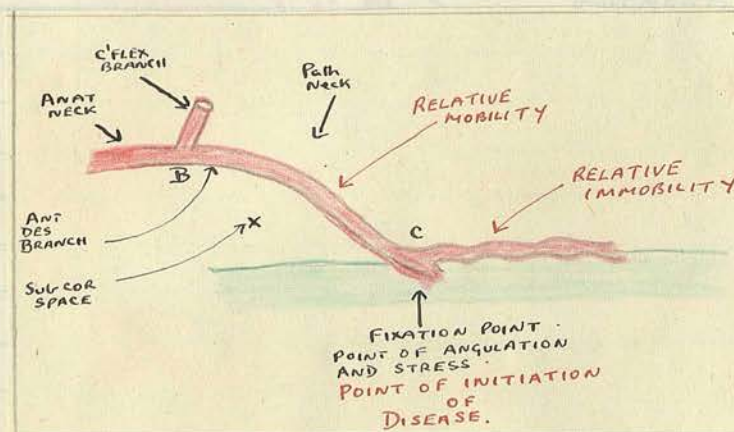


Diagram 2.

If one takes a section at this point of stress and angulation one sees often much pathological change like that in the micro-photograph. (*Plate one. Page 3.*)

In this section taken from a point C of a diseased artery the angulation is seen with much calcification and rupture almost through of the coats of the artery. What are the forces that take part in causing stresses in the left coronary artery which plays so important a part in the welfare of the heart, and which embryologically is only an enlarged aortic vasa vasorum?

I have outlined these stress points in diagram 3. *Page. 6.* Let us consider them one by one with the help of the diagram.

DIAGRAM OF LEFT CORONARY STRESS POINTS.

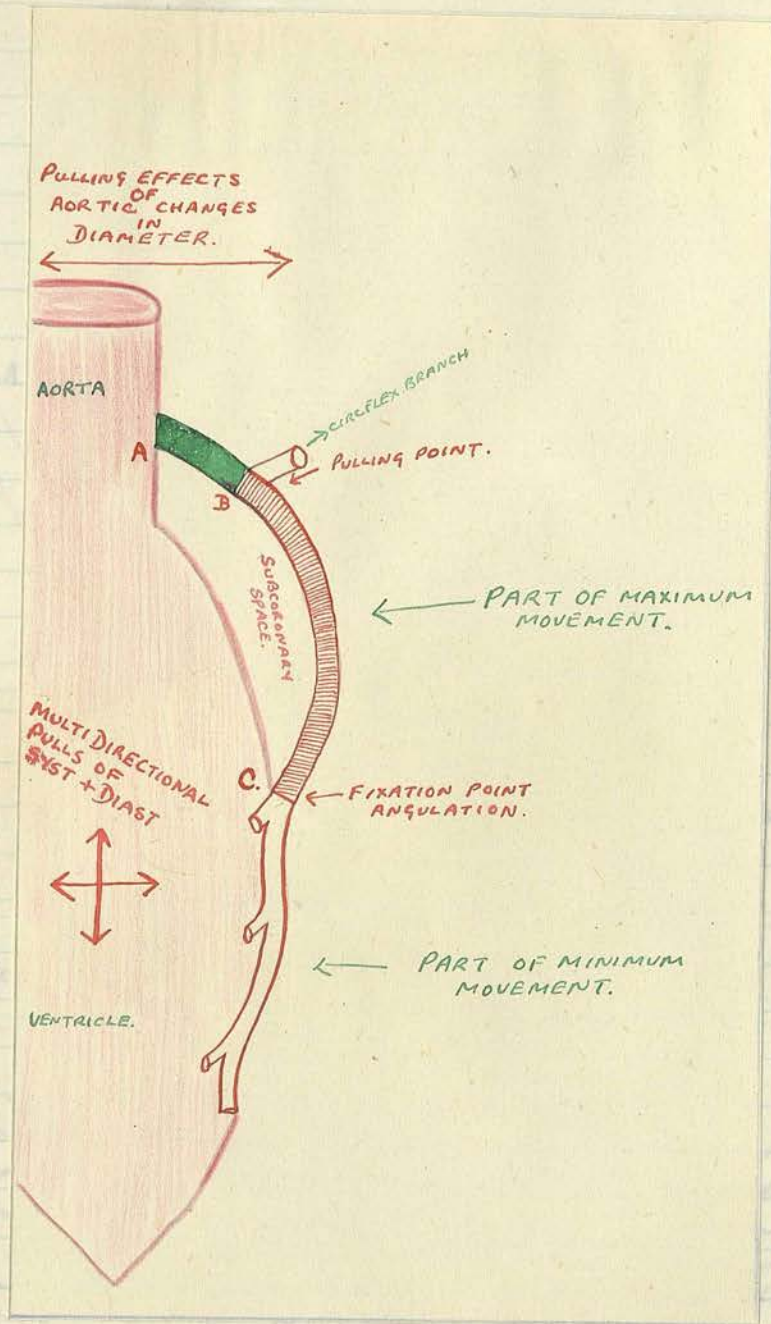


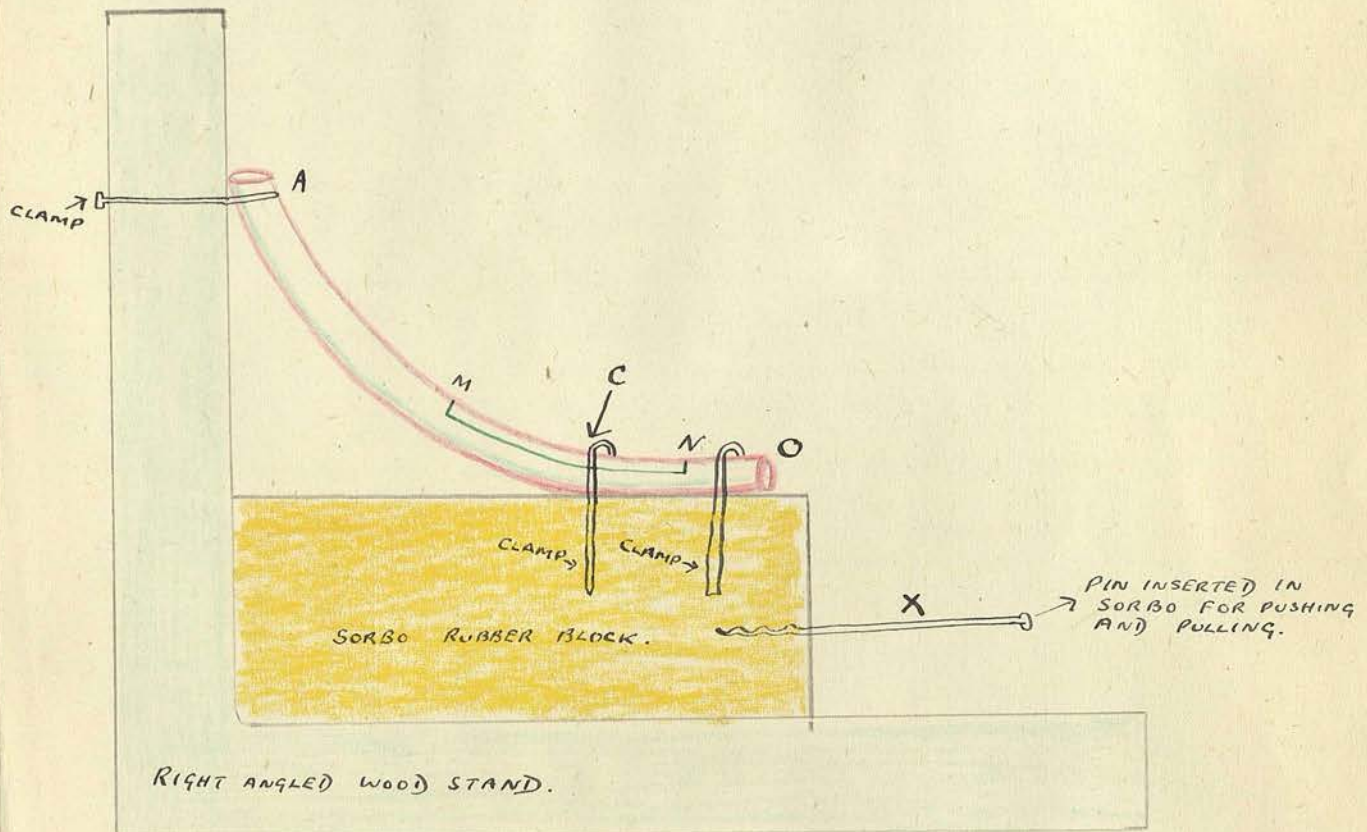
Diagram 3



ANATOMICAL NECK.



PATHOLOGICAL NECK.



A length of rubber tubing is clamped at each end to a right angled wooden stand in the above manner. At A one clamp is placed — at O two clamps to get the tube flat on the rubber. Ordinary sorbo rubber is fastened firmly in the angle of the wood stand with adhesive to form a small rectangular block. At one end the pin X is inserted so that by pulling and pushing it part of the block expands and contracts elastically so point C moves back and forth [C here is analogous to point C on the real artery — the fixation stress point of the descending anterior branch of left coronary]. Ordinary section cutting hot wax is run down the tube and left to form a coating on the floor of the tube. Then the Sorbo is worked back and forth. The piece M-N is cut off the top of the tube as an inspection chamber. The wax along A to C will be in a good state but at C it will be cracked up due to the stress due to disparity of movement at this part.

The left coronary artery is attached to the aorta which expands and contracts with each cycle of the heart, displacing point A backwards and forwards and up and down. The heart body contracts and expands rhythmically displacing point C up and down and laterally. The arch roofing the sub-coronary space is bent and straightened rhythmically. From point C distalwards the descending vessel is moved in and out by changes in the ventricular diameter; but in comparison with the movement at A to C this is small. So point C is a point of stress and constantly varying angulation with disease changes in all cases.

All this happens only where the anatomical neck is short when the descending branch has to complete the arch and form the pathological neck.

Where there is a long anatomical neck the arch is mostly or all formed by the coronary stem or neck, and the descending branch originates on the flat and has no angulation and stress point.

Some idea of the stress formed when such a pathological neck is present can be seen in the performance of a simple experiment described in diagram 4 Page 7. Although in this experiment the curve of the tube is in opposite direction to the coronary curve, the same wax damage is seen when the tube is fixed in the opposite way - a rather more difficult undertaking.

The same kind of pathological strain due to angulation and stress points and branch origins as take place in the coronary has been described as taking place in the aorta and other vessels by the undermentioned writers.

Duguid J.B. J.Path. XXIX, 4. 376. 1926.

Allbutt. Diseases Heart. Arteries. 1915.

Willius F.A. Proc Mayo Clinic. Vol 17. 20 1942.

Plesch J. Heart. Blood Vessels. 1937.

Not only can the coronary be divided from the point of view of short and long necks, but also from the point of view of number of main branches and their general pattern. If all the coronaries one comes across in post mortem examinations are dissected out and mounted on strips and kept preserved for comparison as shown in photographs on pages 19. Plate 8., they can be sorted out into pattern types.

In large mental hospitals ~~men~~ members of the same family die, and I have noticed ~~the~~ familial patterns, which are the same almost in every respect.

A good pattern or a bad pattern may be inherited like other anatomical characteristics, thus explaining the death from coronary thrombosis of several members of a family. More than once I have seen father and son die of coronary thrombosis at the same age and at the same point of the artery. Levine records this too but can give no cause. Inheritance of similar short anatomical necked patterns supply the explanation.

Dissections of the coronaries are much better for study than injection methods, because there may be lack of clearness due to overlapping.

So far all my coronary death cases have been where there were short coronary anatomical necks.

The right coronary artery is usually a lesser artery with supply to lesser muscle area, and with its ramifications more gradual than abrupt, and less activity and stress points. Mechanically unfavourable patterns of right coronary vessels can be found with severe pathological consequences.

Dissected arteries can be X rayed in groups as seen in the photographs on pages 20 to 24. These can be used to study and record patterns, and also to study wall thickening and especially the deposit of calcium.

In my experience calcium deposition does not follow any rule, being found in slight and severe pathological changes and in all kinds of patterns. Very often however initial calcification takes place at stress points in the pathological neck.

Let us consider a reported case from the research point of view.

Fatal Coronary Sclerosis

In a Boy of Ten Years

At Autopsy the left descending branch of the coronary artery was blocked for a distance of about an inch, beginning $\frac{1}{2}$ inch from the orifice. Above and below the occlusion were slight atheromatous changes in the intima. Histological examination of the diseased portion of the coronary artery revealed an almost complete occlusion. The intima was considerably thickened and hyalinised and a well organised thrombus occupied almost the whole lumen of the vessel.

There was well-marked cellular activity inside the thrombus. Several plaques of calcium were deposited between intima and media and the surrounding tissues were infiltrated with erythrocytes. The internal elastic layer was disrupted and completely absent in parts. No other abnormalities were found in the arterial system. A special effort was made to study the boy's family and previous history, but no light could be thrown on the origin of the condition.

(Lancet Nov. 18 1944. P. 659)

In the above case it would have been interesting to have known the pattern, whether it had severe mechanical disability, and one suggests that these cases require such investigation. The other members of the family when they die could be investigated, as it is only by such familial study that the patterns can be classified both from the pathological and the hereditary point of view.

I have endeavoured in this type of research and in what I have written about the coronary arteries to give a new line for study and thought in the elucidation of their pathology.

I enumerate below some general literature on the subject which I have found helpfull in thinking out the problems of coronary disease.

Mechanics of heart and coronaries.

- Muller E.A. Arch Ges Physiol. 243. 336. 1940.
Evans and Starling. J. Physiol. 46. 418. 1913.
Starling. Physiology. 8th Edit. 1941.
Rec Advan Physiology. 5th Edit. 1936.
Howell. Physiology. 14th Edit. 1940.

Diseases and pathology.

- Bramwell and King, Cardiology. 1942.
Recent adv Cardiology. 3d Edit. 1935.
Levine. Clin Heart Disease. 2nd Edit. 1940.
Lewis. Sir T. Dis of Heart. 3d Edit. 1941.
Plesch. J. Physiol, Path. Heart and Vessels. 1937.
Willius. F. A. Cardiac Clinics. 1941.

Papers dealing directly with Coronaries.

- Whitten M.B. Coron Arts. Arch Int Med 45. 383. 1930.
Osler. and McCrae. Mod Med. Vol 4. 449. 1915.
Pardee H.E.B. Dis Coron Arts. Med Clin N. Amer.
4. 1491. 1921 .
Porter W.T. Further researches cor arts.
J. Exper Med I.46. 1896.
Reid W.D. Mech of Angina Pect. Arch Int Med.34.137.1924.
Neuhof S. Clin Card. 1917.
Moritz A.R. Syph Cor Arteritis. Arch Path. II.44. 1931.
Miller J.L. Effect on H of L Cor Obst.
Arch Int Med. 3. 476. 1909.
Mackensie J. Dis Heart. 1913.
McCallum W.G. Pathology. 7th Edit 1942.
Karsner H.T. Path of Endoc. J.A.M.A. 72.387.1919.
Herrick. J.B. Thromb Cor Art. J.A.M.A. 96.411.1931.
Hirschfelder. A.D. Dis H and Aort. 1918.
Benson R.L. Present status Cor Dis.
Arch Path Lab Med. 2.876. 1926.
Aschoff L. Path Anat. Edit 5. 1921.
Boyd. Pathology. 3d Edit 1938.
Lichtwitz. Funct Pathology. 1942.
Muir R. Pathology. 5th Edit. 1942.

The next pages contain additional photographs to illustrate points regarding coronary artery disease.

Summary of this part of the Thesis
and Photographic notes over leaf.

Summary of Observations on the Coronary Arteries

- (1) Attention is drawn and authorities quoted to show the prevalence of disease in the first part of the anterior descending branch of the left coronary artery.
- (2) The normal anatomy of the artery is described.
- (3) By description of experiments and photographs of cases, the cause of disease in the first part of the descending artery is shown to be mechanical.
- (4) Patterns of coronary arteries and their hereditary transmissions are discussed and photographs shown.
- (5) Three anatomical terms are used for the first time
 - (1) The subcoronary space.
 - (2) The anatomical neck of the left coronary artery.
 - (3) The pathological neck of the left coronary artery.
- (6) The sequence of events in Coronary Thrombosis and obstruction being
 - (a) Mechanical disability due to a pattern that can be inherited.
 - (b) Tissue injury due to this.
 - (c) A place of least resistance is created.
 - (d) This place of least resistance can have its pathological state hastened and magnified by prevalent blood states such as toxins cholesterol etc.

From the study of a paper just published, the nerves play little part in the causation of coronary thrombosis.

Excitement of cardiac vagus fibres induced in dogs for two hours daily by intravenous infusion of "Posterior Pituitary Extract for periods from three months to four years failed to produce coronary thrombosis. So chronic vagal irritation does not produce.

Ettinger G.H. Canad Med Assoc Jour.
Jan 1945. Vol 52. Page 59.

Photographic Notes

(1) Photomicrograph on Page

Camera = Leica with micro-ibso-attachment.

Film = Kodak Panatomic x.

Filter = Green.

Exposure = 1 second.

Magnification = X 350

Developer = Kodatol fine grain D.K. 20

(2) All other photographs taken by Leica.

ANAT. NECK.



UNDISECTED OPEN.

CUT OPEN UPPER PART.

This is a specimen of a left coronary artery showing a "short necked" type with at X a tendency to coat weakness and kinking. In the angle between the aorta and the circumflex branch is seen some small branches to great vessels, valves etc. which also play a part as "mooring ropes" with a tugging effect.

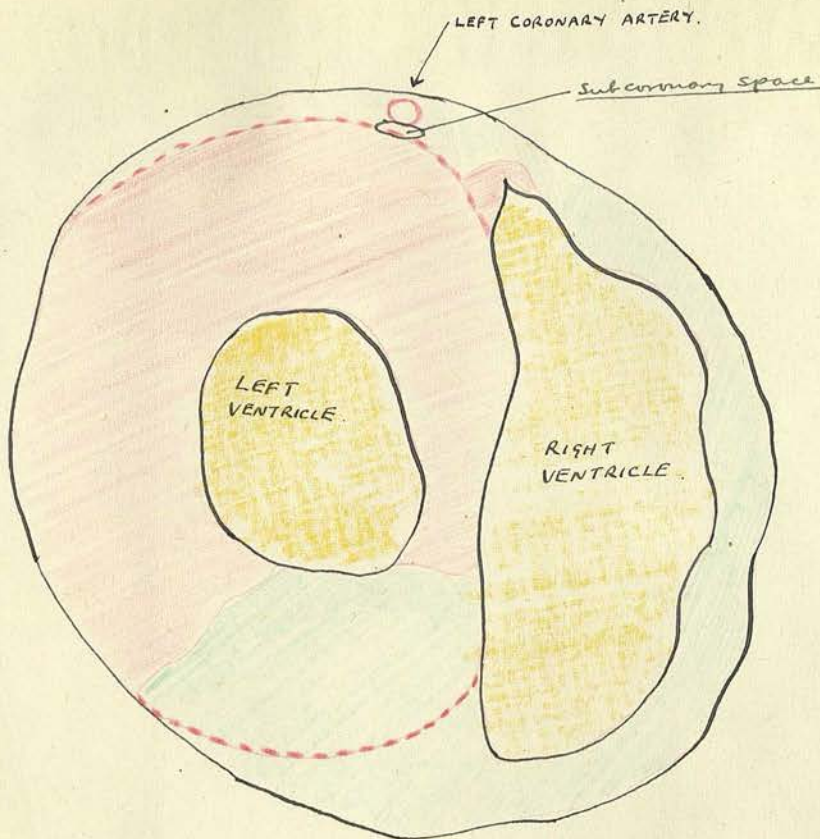
X RAY PHOTOGRAPH OF LEFT CORONARY ARTERY.



POINT X
WITH SMALL CALCIFICATION.

Pathological Neck.
No calcium but
pathological changes
found. This person
did not die of
coronary disease.
An abdominal
emergency ended
his life before
the coronary disease
had time to develop
grossly.

This is the same artery as on the previous page showing the short neck (Anat)
with calcium forming at the stress areas.



TRANSVERSE SECTION THROUGH VENTRICLES WITH LEFT CORONARY ARTERY
INJECTED TO SHOW ITS AREA OF SUPPLY.



LEFT CORONARY SUPPLY



RIGHT CORONARY SUPPLY.

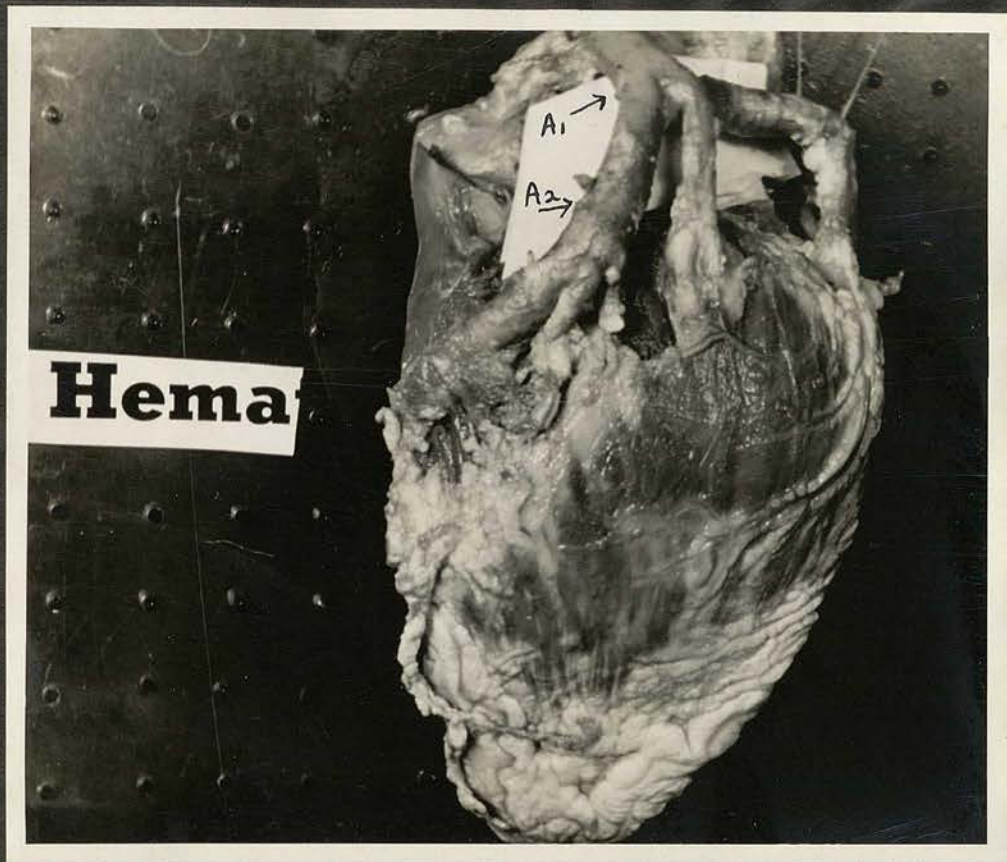


BOUNDARY OF LEFT VENTRICLE MUSCLE.

THIS DIAGRAM SHOWS

an average area of supply of left coronary artery. It supplies and has its ramifications in the most active and muscular ventricle and operates in the sphere of greatest stress. In a transverse section also it is seen that the artery in its first part especially has very little support and there is a distinct space between the neck and the solid heart muscle. (The sub coronary space).

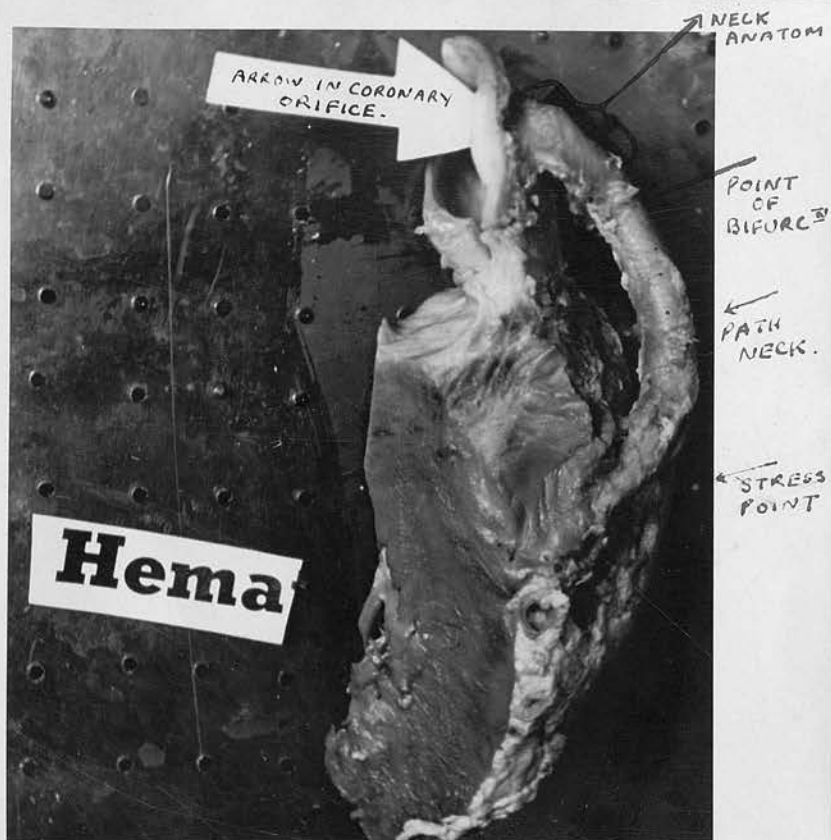
PHOTOGRAPH OF HEART WITH LEFT CORONARY CLEARED AT ITS ORIGIN.



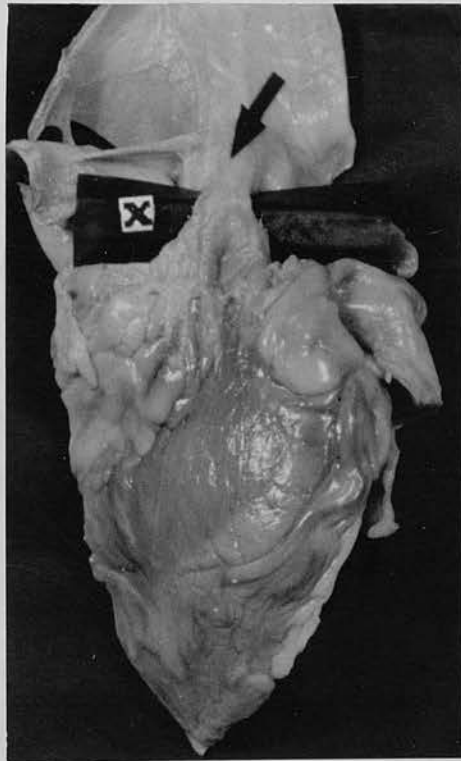
This photograph shows piece of white cardboard thrust under origin of left coronary artery to show the space under it which is usually full of fatty and other weak tissue. (Subcoronary space). This is a short neck type with three large branches in its pattern which dip firmly into muscle lower down and form a stress producing fixation point.

This pattern was found in the other two members of the family of the man to whom this heart belonged, showing a tendency to hereditary transmission of patterns.

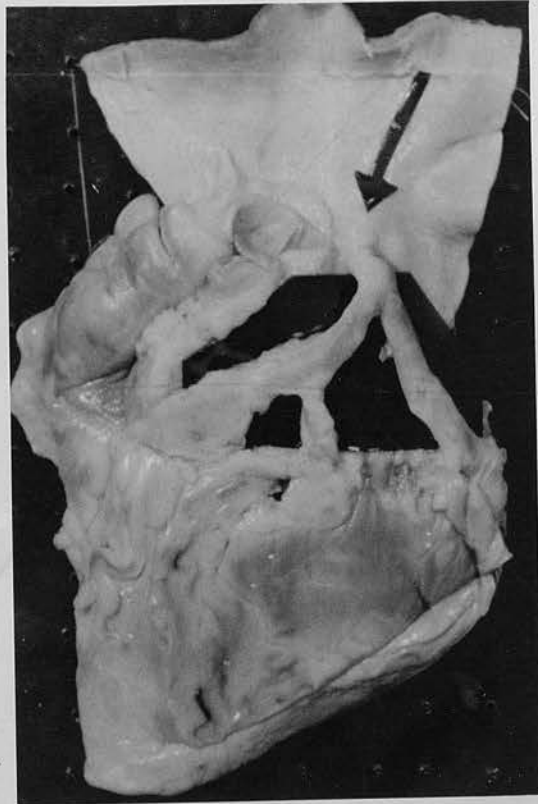
Sections at A₂ showed wear and tear with occlusive changes. Mechanically and clinically the "short necked type" seems a danger pattern. Arrow at A₁ shows point where anatomical neck above and pathological neck below meet.



This photograph shows a lateral view of the same artery. There is a marked gap under the neck and the branches before they take from hold. This side view shows well the sub-coronary space.



This is the photograph of another type of short necked left coronary artery which had atherosclerotic changes. A roll of black paper X was thrust beneath to show unsupported neck. (The sub coronary space). Black arrow points to anatomical neck.



This is another short necked type of left coronary artery which showed atherosclerotic changes. Instead of having a "triple" bifurcation at neck like the previous ones - this and the last artery described have just two large branches - quite a common pattern found amongst studies of several hundred cases. The black paper shows the "sub-coronary space". This on investigation was a "short necked type".



A LONG
NECKS

← SHORT
NECKS.



B SOME
SHORT NECKS.

Typical
X Long
neck.

X X
Short necks.

X RAY PHOTOGRAPHS TO SHOW PATTERNS OF LEFT CORONARY ARTERIES

A ↓ B ↓



Calcium deposition does not seem to follow any pattern
These are all comparatively long necked type
A and B belonged to same family and showed familial similarity of pattern.

XRAY PHOTOGRAPHS OF LEFT CORONARY ARTERIES.

Page 21



F1

F1

F2

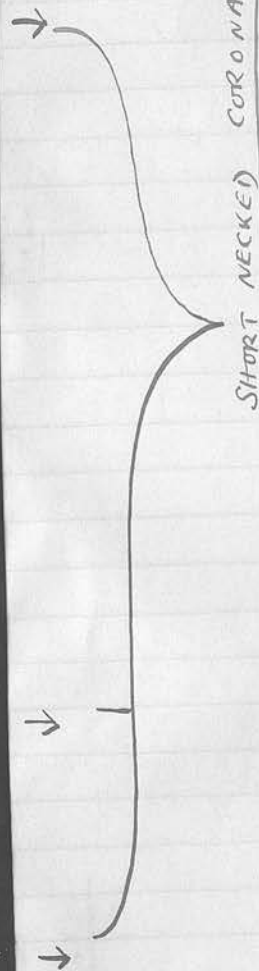
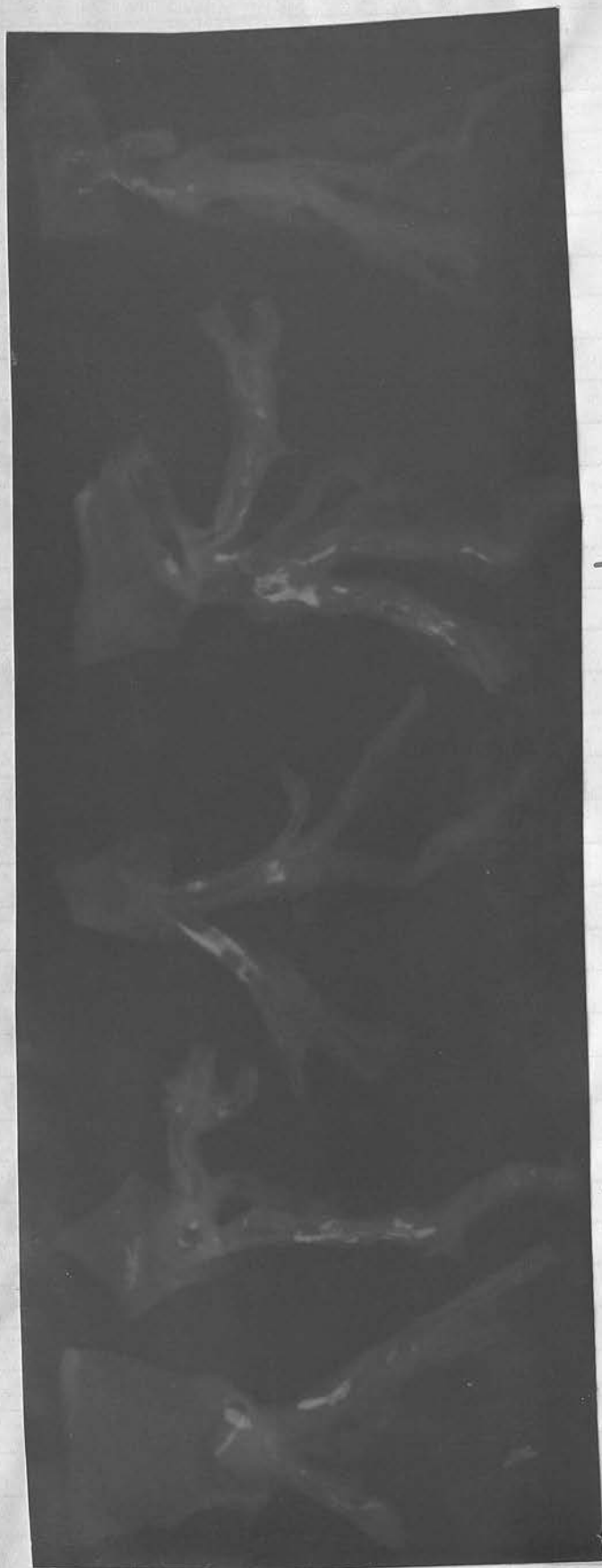
F1, F1 and F2 belonged to the same family, showing familial likeness
Only one artery calcified — calcification does not seem
to follow any special pattern.

XRAY PHOTOGRAPH OF LEFT CORONARY ARTERIES
SHOWING PATTERNS.



Same family
slut mixed coronaries

X RAY PHOTOS OF LEFT CORONARY ARTERIES SHOWING PATTERNS.



SHORT NECKED CORONARIES.

X RAY PHOTOS OF LEFT CORONARY ARTERIES SHOWING PATTERNS.



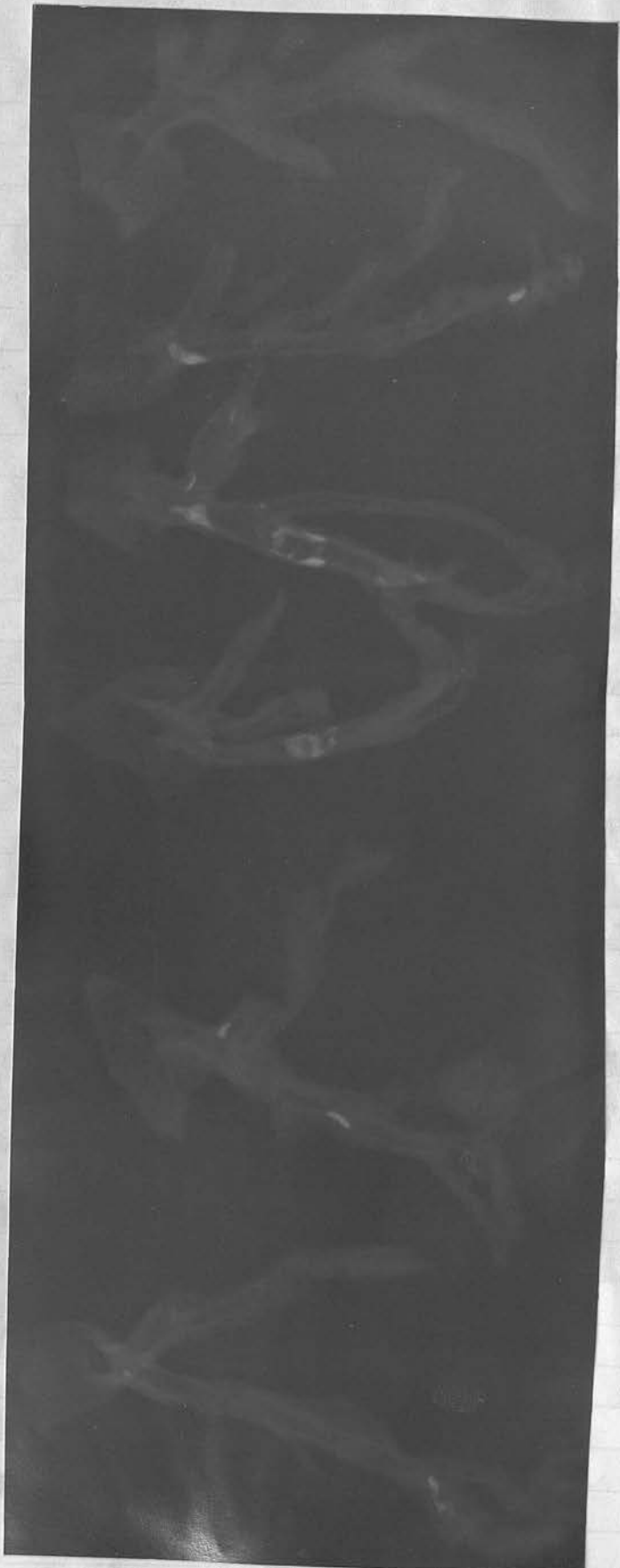
↓
SHORT
NECK.

↓
LONG
NECK.

↓
SHORT
NECK.

↓
LONG
NECK.

XRAY PHOTOS OF LEFT CORONARIES SHOWING PATTERNS.



TYPES OF SHORT NECKS.

PHOTOGRAPH OF ANEURYSM OF LEFT VENTRICLE.

Page 26



Heart cut in two halves.

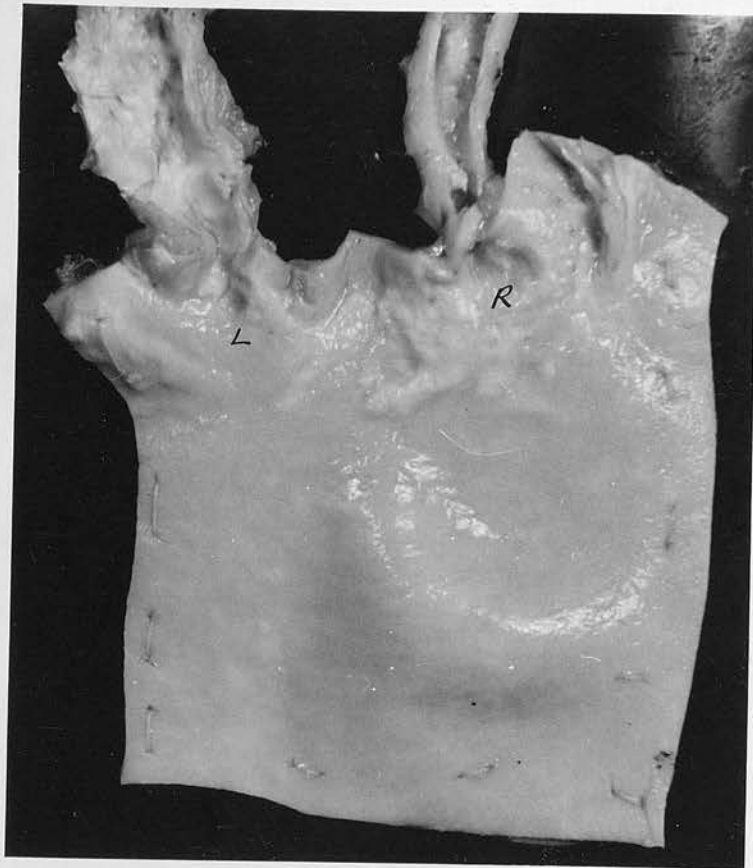


In this specimen which had a short neck coronary there were obstructive changes which had caused the aneurysm. As mentioned before in the pattern photographs Calcium deposit does not follow any rule — many diseased arteries had no calcium demonstrable by X Rays. In this case as seen by following X Ray no Calcium could be seen.

XRAY PHOTO OF PREVIOUS
SPECIMEN (BECK) TO SHOW
absence of demonstrable
calcium deposit in spite
of much coronary disease.

Thick wall of
aneurysm





Photograph of opened coronary with
bilateral coronary disease.

The left cor (L) was of the short anatomical
necked type and the disease caused death.
The circumflex branch of L has been cut
away at its origin.